ARIZONA MEDICINE

Journal of ARIZONA MEDICAL ASSOCIATION

VOL. 6, NO. 2 FEBRUARY, 1949

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Published monthly by the Arizona Medical Association. Business office at 418 Heard Building, Phoenix, Arizona. Subscription $3.00 a year, single copy 25c. Entered as second class matter March 1, 1921, at Postoffice at Phoenix, Arizona, Act of March 3, 1879.

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[2] Spies, T.D., and Collins, H.S.: Observation on Aging in Nutritionally Deficient Persons, J. Gerontol. *1*:33 (Jan.) 1946.

[3] Stieglitz, E.J.: Therapy of the Aged, M. Ann. District of Columbia *17*:197 (Apr.) 1948.

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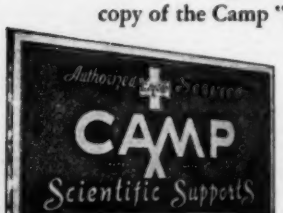

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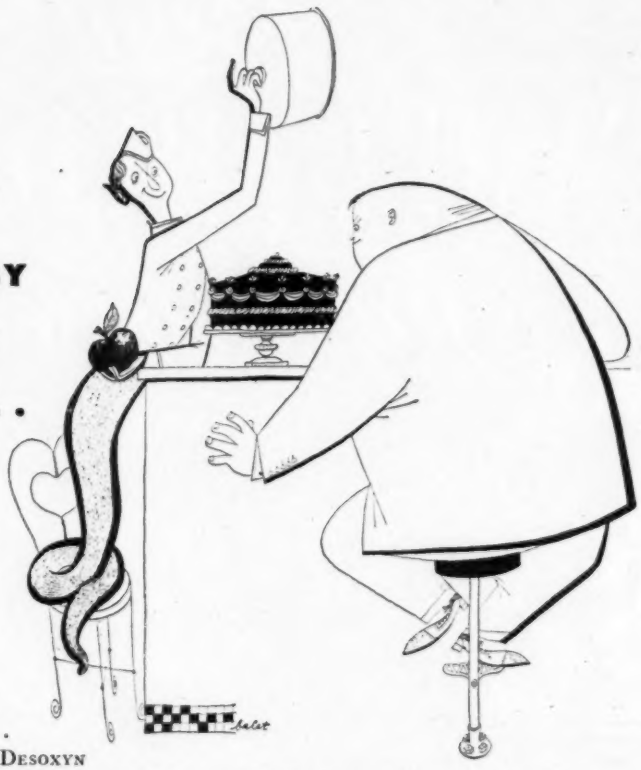

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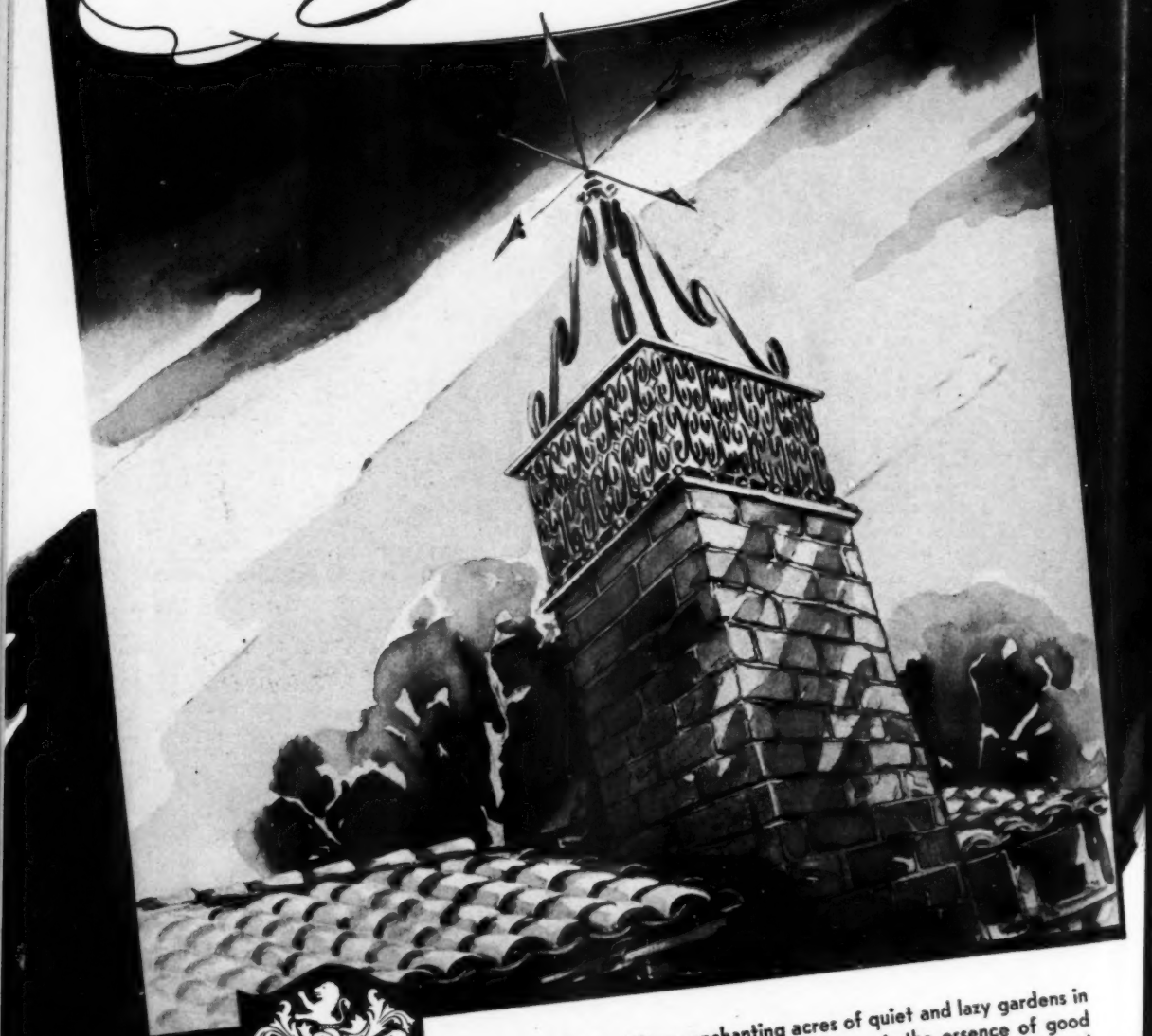

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ARIZONA MEDICINE

Journal of ARIZONA MEDICAL ASSOCIATION

VOL. 6, NO. 2 FEBRUARY, 1949

ACUTE COMPLICATIONS OF FRACTURES

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IF the possibilities of acute complications of fractures are understood, the treatment of these complications frequently is obvious. If the mode of production is known, the dangers of producing these complications while treating fractures is lessened. It sometimes helps to be able to see at a glance the extensiveness of the problem at hand. The acute complications of fractures are listed in table 1.

On occasions the complications of a fracture assume such importance to limb and function that the fracture itself is best disregarded and attention focused on treatment of the complication. Fractures in themselves do not require emergency treatment. If the patient is comfortable, they can be reduced today, tomorrow or next week, but the complications may require emergency treatment.

Lesions of the Skin

The skin overlying a fracture frequently is damaged. There may be minor brush burns or superficially embedded foreign material, such as cinders or gravel. Scrubbing, debridement and a petrolatum gauze dressing will usually suffice. Within a few hours after a fracture, considerable edema may develop in the extremity. This may be followed within twenty-four hours by the formation of huge blebs. If a reduction is secured and the limb is encased, the reduction is frequently lost as the edema subsides. It is better to elevate the extremity until the edema has subsided if it occurs. The blebs can be removed, and a petrolatum gauze dressing can be applied at the time of the reduction.

If the break extends through the dermis, the fracture must be considered to be compound and the depth of the wound must be inspected. After the wound has been cleansed thoroughly, it usually can be closed. If there is any doubt about whether the wound should be closed immediately, it should be loosely packed open. The advisability of using internal fixation in cases of compound fracture is debatable. I use this method of treatment and feel that it is safe if the wound has been prepared properly by thorough debridement. This method of treatment was used prior to the advent of chemotherapy. It is safer now than it was before chemotherapy was available, but one should not depend entirely on chemotherapy to prevent complications. Good surgical principles must be followed.

If the loss of skin has been sufficient to prevent closure of the wound, primary skin grafting may be employed. A muscle bed usually can be prepared for the graft. It has been amazing to see split-skin grafts take after they have been applied even over cancellous bone and over tendons. Primary skin grafting frequently will save much time and minimize the disability.

No consideration of lesions of the skin would be complete without mention of decubitus ulcer. In cases in which the patients are elderly persons, this complication may be acute. Overnight is sufficient time for decubitus ulcer to develop, and its development is not always the fault of the nurses. Advice must be given as to how to avoid this complication. The most common sites for decubitus ulcers are well known and they should be inspected daily.

Pressure ulcers are much easier to prevent than they are to treat. Pressure ulcers which are caused by the use of casts and traction are

Read at the Lois Grunow Memorial Clinic Lectures, Phoenix, Arizona, February 19 to 20, 1948.

always the fault of the surgeon. They are preventable by proper application of casts and traction and by careful observation. It is my practice to assume that the complaints of a patient always are justified. This is particularly true in cases in which the patients are children. If a patient complains of pain, burning, numbness and pressure, the surgeon should inspect the site of the symptom even if such inspection necessitates the removal of a cast or a traction apparatus.

Vascular Lesions

If the injury of the soft tissues is extensive and involves the blood vessels, vasospasm may develop. This may be severe enough to cause serious ischemia of the extremity. This vasospasm and ischemia occasionally occur after a tourniquet has been applied for the usual length of time. Such a simple procedure as the application of warm packs or lowering of the involved extremity may relieve the vasospasm immediately. If this does not suffice, other therapeutic procedures should be instituted immediately. Intravenous or intra-arterial administration of ½ to 1 grain of papaverine hydrochloride may be effective. Intravenous administration of as much as 60 cc. of ethyl alcohol in isotonic solution of sodium chloride may prove beneficial when used in combination with papaverine hydrochloride or morphine. Etamon chloride (tetraethylammonium chloride) may be administered intravenously, twice a day, in doses of 100 to 500 mg.; however, its use is not without danger and it should be administered with caution. Parasympathetic block and spinal anesthesia frequently are helpful. Surgical excision of the injured blood vessels may be beneficial in some cases. This procedure proved effective in the recent war in relieving vasospasm in collateral vessels when a major artery was injured.

Venous obstruction may occur in cases in which the injury occurs about the shoulder girdle or pelvis. Thrombosis of the large veins may cause severe edema and swelling of the extremity. Elevation and elastic pressure dressings to facilitate tissue drainage are about the only means at hand to combat this complication. The use of anticoagulants in cases of extensive hemorrhage and damage to the soft tissues is felt to be dangerous.

The axillary artery and vein are occasionally injured by sharp fragments of the clavicle. The axillary artery occasionally may be injured or

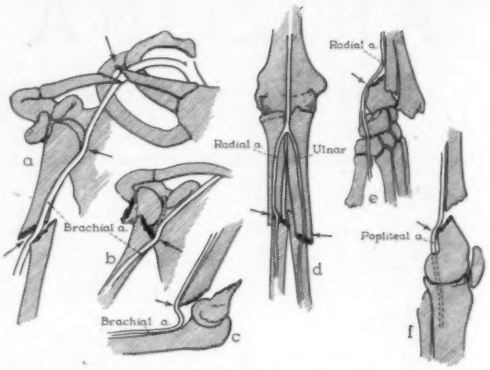

Fig. 1a to f. Most common sites of vascular lesions associated with fractures and dislocations.

ruptured by the sharp edges of the lower fragment of the humerus in cases of fracture-dislocation of the neck of the humerus.

In cases of compound supracondylar fracture in which the patients are children, the distal end of the proximal fragment frequently presents in the cubital fossa at the elbow. I have had to ligate the brachial artery in two cases in which this type of fracture caused irreparable injury to the artery (Fig. 1). Thrombosis must occur rather frequently in cases in which injury occurs in the supracondylar region. Extravasation of blood into this rather tight cubital compartment frequently interferes with the circulation of blood in the forearm. No time should be wasted in releasing the pressure by open operation if it is feared that Volkmann's ischemic paralysis may develop. If this complication occurs, the fracture becomes of secondary importance.

The radial artery occasionally is injured in cases of comminuted fracture of the wrist.

The vessels of the lower extremity are somewhat less vulnerable than those of the upper extremity. The principal point of danger is the popliteal space where, in cases of supracondylar fractures of the femur, the distal fragment may be angulated posteriorly and cause pressure on, or possibly rupture of, the popliteal artery.

If a hematoma develops at the site of arterial rupture, there is always danger of the formation of a false aneurysm. Pulsations of the artery distal to the aneurysm are lost and circulatory embarrassment may be sufficient to cause gangrene. An arteriovenous fistula, that is a true aneurysm may develop if a fragment of bone perforates a vein and an artery. In cases of this type of injury there is danger that gan-

grene may develop. The fistulous tract should be treated surgically. Thrombosis of an injured artery is not uncommon and resection of the involved portion of the artery is the recommended treatment. If injury of a blood vessel causes a false aneurysm, an arteriovenous fistula or thrombosis, there will be a permanent decrease in the flow of arterial blood to the involved extremity. If the resulting ischemia is severe, no time should be lost in performing an appropriate type of sympathectomy.

Volkmann's ischemic paralysis is closely related to the vascular complications which have just been considered. A complete understanding of this complication is not yet at hand. It cannot be considered in detail in this paper. It should be treated immediately and the fracture should be disregarded. Compression of the vessels should be relieved and the circulation should be improved by sympathetic block or sympathectomy.

Where stasis develops in veins, the danger that thrombophlebitis may develop is always present. The presence of thrombophlebitis should be suspected when pain and tenderness develop in the groin or axilla of a limb encased in a cast. The cast should be bivalved and, if the presence of this complication is confirmed, anticoagulant therapy should be instituted. I do not believe that such radical procedures as ligation of the femoral or iliac veins are necessary, but, there may be a place for such procedures in the treatment of this complication.

Lesions of Peripheral Nerves

Fractures or dislocations may be complicated by injury of the peripheral nerves. Injuries of the peripheral nerves may be primary or secondary. Primary injury occurs at the time of the fracture or dislocation. It may be due to the same external force that produced the fracture or dislocation, or it may be caused by impingement of the fragments at the time of the accident or when the fracture or dislocation is reduced. Secondary injury occurs in the course of healing. It is caused by the formation of scar tissue in or about the nerve or by impingement of callus on the nerve.

The injury of the nerve may be of several types. Traction on the nerve because of the displacement or elongation of the bone may cause rather extensive longitudinal damage. Compression or severance of the nerve may be caused by a sharp spicule or by the margins of the fractured bone.

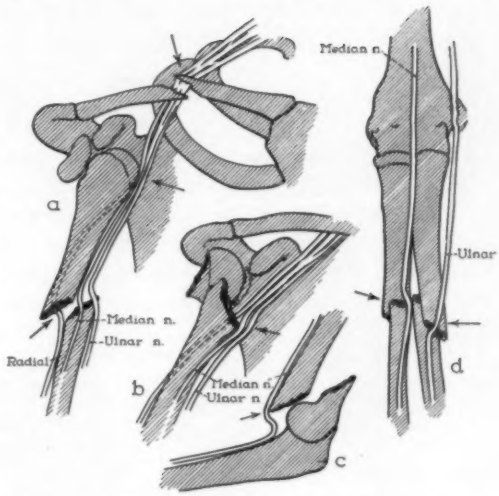

Fig. 2. Most common sites of nerve lesions associated with fractures and dislocations; **a, b, c** and **d,** upper extremity.

The brachial plexus and axillary nerves are the most frequent sites of traction injury. Clinically, it is difficult to distinguish the two types of injuries. If one can demonstrate that the lesion is proximal to the site of fracture by the sensory findings and the paralysis that is present, it may be assumed that a traction injury must have occurred. In cases of this type of injury the prognosis is worse than it is in cases in which the injury was caused by a contusion or laceration.

The most frequent sites of nerve injuries are shown in figure 2. In order of decreasing frequency of involvement they are the radial, ulnar, median, peroneal and sciatic nerves.

Treatment should be tempered by the nerve that is injured and by the site of the lesion in the course of the nerve. When the distance between the lesion and the first motor unit which would regain function is great it may not be wise to wait too long before the nerve is explored. Nerve regeneration is estimated to occur at the rate of 1 mm. per day. When the lesion is complete and expectant treatment is pursued for a long time, much degeneration in the distal portion of the nerve and in the muscle fibers will occur before one can be sure that the lesion is complete. In cases in which surgical repair is employed late, the prognosis for recovery is not as good as it is in cases in which the nerve is repaired early.

Early exploration is best applied in cases in

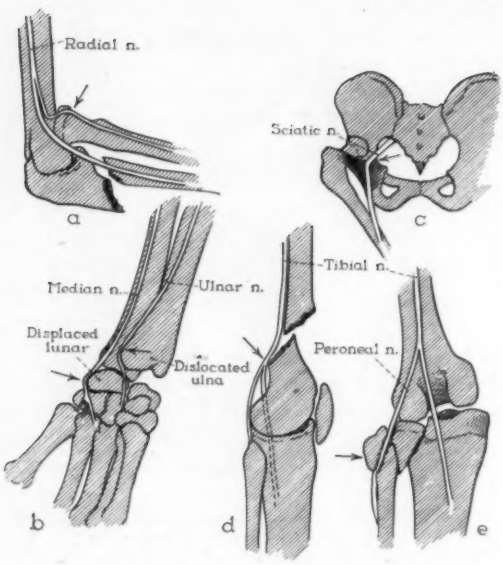

Fig. 3. Most common sites of nerve lesions associated with fractures and dislocations (continued from figure 2); **a** and **b**, upper extremity; **c** to **e**, lower extremity.

which the lesion involves the median or ulnar nerve above the elbow and in cases in which the lesion involves the sciatic nerve. When the distance between the nerve lesion and the first muscle to regain innervation is less than 100 to 130 mm., as is the case of the usual lesion of the radial nerve in the radial groove, expectant treatment can be used.

Electropotential readings of paralyzed muscles may show motor unit action before gross muscle power can be detected and may throw weight against exploration of a nerve when it is felt that recovery should have taken place in the time allotted.

I wish that we were all perfect and that paralysis never occurred as a result of the use of casts or traction. Such paralysis is for the most part avoidable and knowledge of where to exert care to avoid pressure is important. Danger points are shown in figures 2 and 3.

Lesions of Muscles, Tendons and Ligaments

The lesions of the muscles, tendons and ligaments that can occur in cases of fracture are almost too numerous to mention. These structures may be contused, incarcerated or ruptured. When contused, they most frequently undergo spontaneous repair; however, contusions may cause hemorrhage in or about muscles and may cause myositis ossificans or an ossified hematoma. Unless the last two lesions are extensive, they do not require any treatment.

Incarceration of muscles, tendons or ligaments between fragments of bone precludes closed reduction of a fracture. Muscle most frequently is caught between fragments of the shaft of the femur and between fragments of both bones in fractures of the forearm. When such incarceration occurs, open reduction is necessary as this complication is a frequent cause of malunion and nonunion.

Tendons may slip between the fragments of bone in cases of fracture about the joints. Open reduction and accurate replacement are necessary if good function of the joint is to be obtained.

Rupture of muscles, tendons and ligaments is an occasional complication of fractures. Such rupture may occur at the site of fracture and may be the result of direct trauma and strain of the involved region. It may be caused by indirect trauma at a distance from the fracture, such as rupture of the quadriceps ligament of a person who sustains a fracture of the os calcis in a fall with the knee partially flexed. Astute examination is frequently necessary to detect this type of injury. Open operation and repair is the treatment of choice.

Infection

Infection may be a very distressing complication of fractures. It may be exogenous or endogenous in origin. Penicillin has proved to be of great value in the prevention and treatment of infection in cases of fracture. In every case of compound fracture, penicillin should be administered as a prophylactic agent in doses of 300,000 to 1,000,000 units per day, depending on how great the danger of impending infection is believed to be. As an additional measure, I still administer a prophylactic dose of tetanus and gas gangrene antitoxins in all cases of compound fracture.

If one should suspect that gas gangrene or tetanus is developing, a full therapeutic dose of antitoxin should be administered and cultures should be made with material obtained from the wound. Treatment should not be delayed until the cultures become positive; if this is done, the chance of saving the involved limb or the life of the patient may be lost. Surgical incision of the involved tissues is still employed in cases of gas gangrene. I do not think that roentgen therapy is effective in the treatment of this complica-

tion. I have not observed a case of gas gangrene in the past three years. I hope that this apparent decrease in the incidence of this complication is attributable to careful cleansing of the wounds of compound fractures and to the administration of prophylactic doses of penicillin.

Acute Traumatic Atrophy of Bone

Acute traumatic bone atrophy most probably is the result of vasomotor disturbances in the extremity wherein the calcium is washed out of the bone and painful osteoporosis results. Early parasympathetic block and use are the best treatment. The joints should be kept mobilized. All forms of physical therapy frequently prove beneficial.

SUMMARY

The treatment of fractures is not always a simple matter. A thorough understanding of the complications which can occur is important as they are frequently serious when not properly cared for. Lost time, permanent disability and legal entanglement may be avoided if the treatment of fractures is given its proper serious consideration.

TABLE 1

ACUTE COMPLICATIONS OF FRACTURES

I. *Lesions of skin*
- A. Brush burns.
  - 1. Simple.
  - 2. Complicated by imbedded foreign bodies.
- B. Edema and blebs.
- C. Wounds of compound fractures.
- D. Loss of substance.
- E. Decubital ulcers.
- F. Pressure ulcers caused by casts and traction.

II. *Vascular lesions.*
- A. Vasospasm.
- B. Lesions of arteries.
  - 1. Rupture.
  - 2. Thrombosis.
- C. Lesions of veins.
  - 1. Venous obstruction.
  - 2. Thrombophlebitis.
- D. Traumatic aneurysm.
  - 1. False.
  - 2. True.
- E. Volkmann's contracture.

III. *Lesions of peripheral nerves.*
- A. Primary.
  - 1. Traction paralysis.
  - 2. Compression paralysis.
  - 3. Paralysis caused by severance of a nerve.
- B. Secondary.
  - 1. Contractures caused by scars.
  - 2. Compression caused by callus.

IV. *Lesions of muscles, tendons and ligaments.*
- A. Contusions.
  - 1. Diffuse hemorrhage into tissues.
  - 2. Hematomas.
    - a. In muscle.
    - b. Beneath muscle.
    - c. Ossifying hematomas.
      - (1) Myositis ossificans.
- B. Incarceration and associated lacerations.
- C. Rupture.
  - 1. Local at site of fracture.
  - 2. Distant due to force of injury.

V. *Infections.*
- A. Exogenous.
  - 1. Introduced through wounds of compound fractures.
- B. Endogenous.
  - 1. Metastatic.
- C. Bacteriologic types.
  - 1. Pyogenic.
  - 2. Gas gangrene.
  - 3. Tetanus.

VI. *Sudeck's atrophy.*

VAGOTOMY AND THE ULCER PROBLEM

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THE recent and accurate observations of Dragstedt strongly suggest that an excess of unneutralized and undiluted gastric acid is a most important factor in the etiology of peptic ulcers. Pure gastric juice differs from gastric contents in that the latter is a mixture of the former plus saliva, food, mucus, regurgitated duodenal juices and a neutral or faintly alkaline secretion from the pyloric antrum; this was demonstrated in animal experimentation with Hiedenhain or Pavlov pouches. Althonugh pure gastric juice may be detrimental to living tissue, gastric contents are almost entirely inert. In 1814, Brodie observed that the stomachs of dogs filled by intravenous injections of arsenic contained large amounts of mucus and watery fluid; if, however, the vagi were cut prior to the administration of arsenic, the stomach remained empty, therefore, the effect of section of the vagus nerves on gastric secretion is not new. These observations were confirmed by Wood in 1870. Pavlov in 1894, made similar observations on dogs and found that hydrochloric acid was not present following section of the vagus nerves. In 1910, Pavlov emphasized the nervous or neurogenic factor in gastric secretion, which he thought could be completely abolished by vagus section. His deductions were only partially correct, since A. J. Carlson has shown that although the nerve or neurogenic secretion was abolished, there still existed a hormonal (humoral) secretion (gastrin), which is believed to be formed in the intestine and absorbed by the blood stream. When food is present in the stomach, this secretion is formed even though the vagi are cut. The amounts of secretion produced by neurogenic and hormonal stimulation are unknown. Ivy (1925), Lim, Ivy and McCarthy (1925), have made valuable contributions to this subject and have confirmed the work of Pavlov. In 1901, Bickel removed the duodenum in two dogs and reestablished continuity by means of a gastrojejunostomy. The bile and pancreatic ducts were implanted into the anterior abdominal wall. Four and one-half weeks later, an autopsy revealed that one of the animals had developed jejunal ulcers, one of which had perforated producing a peritonitis and death. The rarity of spontaneous ulcer in the dog has been recognized by a number of investigators, therefore, dogs are usually chosen as the laboratory animals for these experiments. In 1911, Exalto noticed that no ulcers developed in dogs in which a gastro-enterostomy was performed, but were frequently noted in those animals in which he did a Roux "Y" operation. He concluded that jejunal ulcers were due to acid gastric juice acting upon the intestinal mucosa. Mann and Williamson described an operation in which the alkaline duodenal contents were shunted into the terminal ileum, and fourteen of sixteen dogs upon which they performed this procedure developed jejunal ulcers. Neuman, Demoor and Deloyers found ulcers in four of seven dogs who had a shunting of their bile into the terminal ileum, and Berg and Jobling produced chronic duodenal ulcerations in thirteen of twenty-three dogs following various types of biliary exclusions.

In 1931, Elman and Hartman produced ulcers in six dogs with pancreatic fistulae and emphasized the importance of pancreatic juice. Berg, however, was able to produce only one ulcer in sixteen dogs with pancreatic fistulae, and no ulcers following total pancreatectomy in eight additional dogs. Kim and Ivy experimenting with neutral gastric mucin noted spontaneous duodenal ulcers in six of ten dogs with biliary fistulae.

Matthews and Dragstedt reviewing the work of Dragsstedt and Vaughn emphasized the importance of the neutralizing effect of saliva, food, mucus and duodenal juices. Ochsner, Gage and Hosoi were able to produce ulcers in 85 per cent of animals in whom gastric pouches were anastomosed to the jejunum, however, if bile was introduced by anastomosing the gallbladder to this pouch the incidence of ulcer dropped to 39 per cent. DeBakey also attempted to answer the perplexing question, namely, which had the greatest protective power, bile, pancreatic juice or succus entericus. He did this by performing gastrojejunostomies with

Read before the Ninth Harlow Brooks Memorial Navajo Clinical Conference, Sage Memorial Hospital, Ganado, Arizona, August 30, 1948. From the Departments of Surgery, University of Illinois, Cook County Graduate School of Medicine, Cook County Hospital, American Hospital and Alexian Brothers Hospital.

Von Eiselsberg pyloric exclusions, and then excluded bile and pancreatic juice separately and collectively. He found with pyloric exclusion alone, 50 per cent of the animals developed ulcers; with pancreatic juice excluded, 70 per cent; with bile excluded, 90 per cent; with both juices excluded, 100 per cent. He concluded that bile was of the greatest importance in preventing jejunal ulcer formation, pancreatic juice next and succus entericus least. In 1941, Varco, Code, Walpole and Wangensteen produced increased gastric secretion and ulcers by intramuscular injection of histamine-beeswax mixture in dogs.

In 1925, McCrea, after reviewing and studying the subject came to the following conclusions: unilateral vagotomy did not affect the motor activity of the stomach and bilateral vagotomy merely decreased the initial emptying time. Section of the vagi eliminated the psychic secretion, but the chemical phase of secretion still persisted. In 1929, Hartzell reported that following transthoracic vagotomies on dogs, complete section of the nerves induced a decrease in acid secretion; transabdominal vagus section showed no constant decrease in the height of acid If the vagotomy was incomplete, no decrease in acid secretion resulted.

In 1930, Dragstedt and Ellis, in an attempt to secure information concerning the relative importance of the vagus nerves in the secretion of gastric juice, performed an experiment with dogs in whom they isolated the stomach (nerve and blood supply intact); intestinal continuity was made possible by an esophagojejunostomy. A gold plated cannula placed in these stomachs collected the secretions. After eating, the isolated stomach secreted from 1,000 to 2,500 cc. of highly acid gastric juice in a twenty-four hour period. The animals would have to be kept alive by supplying them with chlorides, since they would "secrete themselves to death." Those dogs which were kept alive developed ulcers. If the vagi were cut, the acidity and amount of secretion immediately dropped to 300 to 600 cc. per twenty-four hours, and ulcers did not develop. These theoretical considerations led Dragstedt to attempt section of the vagus nerves as a treatment for peptic ulcer.

In 1914, Exner and Schwarzmann performed subdiaphragmatic resection of the vagus in patients suffering with gastric crises, and by adding a gastrojejunostomy they attempted to avert the effects of atony, dilatation and pylorospasm.

In 1920, Stierlin was the first to suggest an operation on the nerve apparatus of the stomach for ulcer cases in humans. His operation consisted of a circular incision through the muscularis and to the submucosa on the upper part of the corpus of the stomach; this incision was then sutured together. By this procedure, he proposed to cut the nerves, but Stierlin's circular myotomy failed because of an erroneous conception of the anatomy of the vagus nerves. Latarjet in 1921-1922, devised an operation, vagotomy plus gastro-enterostomy, in which he attempted to remove sensation, diminish the frequency and intensity of the contractions, and to diminish acidity. Schiassi in 1927, also attempted to cure ulcers by nerve section, and obtained favorable results in a group of twenty-five patients. Alvarez (1928) lowered gastric acidity following experimental vagotomy.

In 1938, Winkelstein and Berg reported that vagotomy alone or in combination with other surgical procedures seemed highly desirable in the therapy of peptic ulcer. In 1944, Weinstein, Colp et al, while writing on vagotomy in the therapy of peptic ulcers, stated that "in none of the cases was clean cut evidence of beneficial therapeutic effect obtained."

In 1932, Cushing stressed the importance of the part played by the central nervous system on the pathogenesis of ulcers. In this modern era of psychosomatic medicine, the importance of nervous tension, emotion, stress, strain, responsibility, competitive effort and frustration have all been emphasized. Winkelstein has presented evidence that the hypersecretion of gastric juice in ulcer patients is of nervous origin, and that subphrenic section of the anterior vagus nerve together with partial gastrectomy or gastroenterostomy lessens ulcer recurrences. The central nervous system may cause ulcers by producing a hypertonus in both the secretory and motor fibers of the vagus, hence, severing them seems logical. Both hypermotility and hypersecretion are abolished by complete section of the vagal fibers, and the secretion and acidity tend to return to normal. Between meals, when there is no buffer action of food, hypersecretion and hyperacidity may prove dangerous. At the present time, Dragstedt has obtained beneficial effects with vagotomy, and to date has reported no ulcer recurrences, however, he states that "a longer period of observation and a more exten-

sive series of patients are obviously necessary to provide final answers."

ANATOMY

Complete severance of the vagus nerves is necessary to successfully correct hypersecretion and hypermotility. To accomplish this it is necessary to thoroughly understand the anatomy. Unfortunately, there are marked contradictions in our leading anatomy text books regarding the vagi. This is probably due to the fact that erroneous information was obtained from the works of Hirschfeld-Leveille, and Kollman in the middle of the nineteenth century, and many of our modern textbooks have obtained their information from these sources.

E. Perman provided the most reliable information on this subject. In 1935, he performed a series of painstaking and accurate dissections dealing with the vagi. His conclusions are interesting, instructive and apparently accurate. He states that both vagi reach the stomach through the lesser omentum, and that they divide into branches without the formation of a plexus of intertwining fibers. He demonstrated that anastomoses exist between the various branches, but they do not lose their individuality as would be the case in a nerve plexus. The *left vagus nerve* sends branches to the liver and to the anterior wall of the stomach. Those traveling to the liver turn to the right, pass through the lesser omentum and to the porta hepatis. The gastric branches divide into several smaller nerves, which spread over the anterior wall and can be followed to the greater curvature. The *right vagus nerve* is arranged in a similar manner; one part to the celiac plexus, and the remainder supply the posterior wall of the stomach. Perman was unable to follow branches of either the left or right vagus into the greater omentum.

Recent studies dealing with the subject of the nerve supply to the stomach are also quite contradictory. Bradley, Small, Wilson and Walters examined one hundred and eleven cadavers, and concluded that there was a great variation in nerve pattern from the cardiopulmonary plexus to the stomach, but the nerves were very constant in course and distribution after reaching the stomach. They divided the nerve patterns into four groups. Miller and Davis studied the vagus nerves below the level of the pulmonary plexus in thirteen cadavers. They state that in only three instances were the nerve arrangements comparable to those described in the text books as "normal", and the remaining ten showed marked variations.

Transabdominal vs. Transthoracic Vagus Nerve Section (Vagotomy)

Walters et al state that in 90 per cent of cases a transabdominal subdiaphragmatic approach will allow as nearly a complete division of the gastric nerves as the transthoracic approach. Miller and Davis, however, are of the opinion that the transthoracic approach is the one of choice.

Each of the approaches have their advantages and disadvantages. The *transthoracic approach* has the following advantages:

(1) Good exposure.
(2) More complete sectioning of the nerves.

Disadvantages:

(1) The lesion cannot be examined (possible malignancy).
(2) Pyloric obstruction cannot be corrected.
(3) Post vagotomy gastric atony cannot be corrected.
(4) Postoperative pain in the incision and costovertebral joint may result.
(5) Gastro-enterostomy is a difficult and unsatisfactory procedure when performed as a secondary operation upon a dilated and atonic stomach.
(6) Intrathoracic complications may result.

Keeping the above facts in mind, it becomes apparent that each case will present a problem unto itself, and that it will be up to the surgeon's judgment and evaluation to decide which of the two approaches is preferable. For example: it is still a fact that every gastric carcinoma is preceded in from 5 to 10 per cent of instances by an ulcer. Such lesions, therefore, should be exposed, removed and examined, whether or not vagus nerve section is performed. It would be a gross error to believe that one has done a complete bilateral vagus section to learn later that some of the vagal fibers have remained intact and a gastric carcinoma developed. The transthoracic approach, on the other hand, is indicated for duodenal ulcers, especially if a previous gastro-enterostomy has been done; this approach is also excellent for anastomotic ulcers if it is deemed unnecessary to remove the ulcer. At present, the transabdominal route is favored for the reasons cited above, and especially since a gastro-enterostomy may be done simultaneously to compensate for the atony which may result following the section of the nerves. Such

atony is no true paralysis, but may result in an acute dilatation of the stomach brought about by the release of the hypertonicity plus swallowed air, saliva and mucus. In our series, eleven cases were done transthoracically and the remaining seventy-six were done transabdominally. The author has also performed vagotomy for cases of chronic, non-specific, ulcerative colitis with dramatic and most encouraging results; however, no definite conclusions can be drawn at this time since a longer period of observation is necessary.

SURGICAL TECHNIQUE

Transabdominal Vagotomy:

A long incision is necessary to obtain proper exposure. We utilize a left muscle-splitting rectus incision which commences in the angle between the xiphoid and left costal cartilage and extends one or two inches below the umbilicus. The peritoneal cavity is entered and the left triangular ligament of the liver is exposed and made taut. This bloodless fold is incised and it then becomes possible to retract the left lobe of the liver to the right. The peritoneum covering the lower end of the esophagus is incised and a finger or scissors is placed into the posterior mediastinum to mobilize the esophagus so that its lower three or four inches can be delivered into the abdomen. The nerves are treated in much the same way as will be described in the transthoracic procedure; at times it is possible to isolate and ligate both vagi on the left side. I have observed that the left (anterior) vagus has a tendency to hug the esophagus and the right (posterior) travels a slight distance away from it; therefore, it is easier to feel and identify the latter. At the conclusion of the operation, the left lobe of the liver is permitted to fall into place; it has been found unnecessary to resuture the severed left triangular ligament. That this structure does not require suturing has been all too well revealed to the author at a postmortem examination performed two weeks following vagus nerve sectioning (case reported below). The left lobe of the liver was found firmly fixed in place. An anterior gastro-enterostomy is usually done following vagus nerve section. The suture material used in this entire series was spool cotton, the only exception being, the use of catgut in the through and through suture when a gastro-enterostomy was performed. These patients are placed upon no postoperative dietary restrictions whatsoever, and early ambulation is encouraged.

Transthoracic Vagotomy:

General anesthesia with positive pressure is used, and an incision is made over the seventh or eighth rib on the left side. A large section of the rib is resected, the pleural cavity entered, and after dividing the inferior pulmonary ligament, the left lung is retracted upward. Some prefer to enter the pleural cavity through an interspace rather than resect a rib. Recognition of the so-called "esophageal triangle" aids in rapidly locating the lower end of the esophagus. This triangle is bounded in front by the heart, behind by the descending aorta, and below by the diaphragm. The pleura covering the triangle is incised and the esophagus identified and mobilized. The main vagus trunks are recognized by touch rather than sight. The nerves feel like taut cords which can readily be differentiated from the more yielding muscle of the esophagus. The fibers are assembled by finger dissection into two main trunks, comprising the right and left vagi which are ligated and divided. Some prefer to take a section of the nerve, but we have not done this routinely. In addition to the two trunks, small vagal fibers penetrate the esophageal wall; these must be combed off the esophagus and severed. The esophagus is replaced in its bed, the overlying pleura sutured and the chest closed without drainage. The patients are encouraged to get out of bed the first or second postoperative day.

POSTOPERATIVE COMPLICATIONS

A postoperative gastric atony, not paralysis, may result. Plus a vagus and sympathetic nerve supply, the stomach has a peripheral nerve supply which gives it an automatism. In general, vagal fibers are augmentors and the sympathetic fibers decrease tonus. Post-vagotomy atony is believed to be due to a transient increased pyloric tonus; swallowed air and saliva may aggravate the condition so that an acute gastric dilatation results. To prevent this, constant Wangensteen suction is instituted for three to five days postoperatively. This is continued until all tendency to develop nausea or vomiting is relieved. Certain medicaments, especially those of the choline group, show some promise in the treatment of gastric atony; time will prove their efficiency. A certain number of patients have a tendency to contract diarrhea, but

this has not proved annoying and has been only transient. Three patients in my series stated that a very resistant and annoying chronic constipation was completely relieved after vagotomy. Foul smelling belching and emesis may result; we have not noticed these complaints. In this series no esophageal spasm was noted.

One of Dragstedt's patients developed a post-vagotomy obstruction, which was thought to be due to the vagus nerve section. However, upon reoperation by Warren H. Cole, this was found to be due to cicatricial stenosis caused by a healing ulcer.

To obtain complete relief and successful results, it is imperative that all vagal fibers be severed. Many tests have been devised to determine whether or not this has been accomplished, but the two most commonly used are the sham meal and insulin tests. Since insulin shock complications are always a possibility with the insulin test, it is essential to have a doctor present for the immediate administration of glucose.

That it is possible to overlook fibers of the vagus nerve seems to be agreed. Dragstedt states that in his first fifty transthoracic vagus nerve sections, three incomplete vagotomies resulted. Hollander reports that in a series of twenty-one cases, forty-eight per cent proved to be incomplete with the insulin test. It must be remembered, however, that gastro-enterostomy or gastrectomy also reduce gastric acidity, hence, when these tests are performed in connection with vagotomy, it may be difficult to tell which procedure is responsible for the resulting achlorhydria.

PERSONAL OBSERVATIONS

To date the author has performed eighty-seven vagotomies in the past two and one-half years; three deaths are recorded in this series, none of which can be directly attributed to the operative procedure. The first of these patients developed a gastric dilatation. This was believed to be due to the bilateral vagotomy, but postmortem examination revealed that the main trunk of the right (posterior) vagus was completely missed and remained intact. A massive left lobar pneumonia accounted for the demise. The second death was in a patient who had a gastro-enterostomy seventeen years prior to entrance. He entered with a diagnosis of a marginal ulcer. A degastro-enterostomy was performed followed by a subtotal gastric resection. Six weeks later, he was readmitted to the hospital with violent pain. Exploration revealed three new acute jejunal anastomotic ulcers. A more radical resection was done and transabdominal bilateral vagotomy added. He obtained immediate relief and left the hospital in a state of profound shock with signs of a generalized peritonitis. He expired and postmortem examination revealed a ruptured jejunal ulcer. The third patient expired forty-eight hours following a bilateral transabdominal vagus section and anterior gastrojejunostomy. Shortly after surgery she developed a shock-like syndrome from which condition she did not recover. Permission for autopsy could not be obtained, hence, no conclusions can be drawn. When analysing these three patients, the immediate cause of death could not be blamed directly to the vagus nerve section.

Weeks, Ryan and Van Hoy reported two deaths associated with supradiaphragmatic vagotomy, but since one of these was associated with a bilateral thoracolumbar sympathectomy, and the second patient had a known methyl alcohol poisoning, and at that only had the right vagus sectioned, it is difficult to see why the vagotomy per se should be blamed.

In our remaining eighty-four cases, the results have been most encouraging. It is indeed gratifying to note the immediate relief obtained following vagotomy, and the ability of the patient to again enjoy a full meal and lead a normal life. Moore et al recently reported a series of forty patients upon whom he performed resection of the vagus nerves. The authors stated that their clinical results were good and that healing of the ulcer was the most uniform feature; transient undesirable side effects were found in about 10 per cent of the patients.

What the future holds for the functions of the liver, pancreas, kidney and intestinal tract following vagus nerve section cannot be stated at this time. However, since the author's last report he is still as enthusiastic about the procedure. Although these pros and cons cannot be definitely answered, the questions should be approached and followed with an unbiased attitude. Whether or not the vagus nerves will regenerate can only be proven by careful "follow-ups" of these patients.

SUMMARY

1. A personal series of eighty-seven cases is reported and three deaths in this series evaluated.

2. The transabdominal approach has distinct advantages in that the lesion may be inspected and a gastro-enterostomy performed to compensate for obstructive or atonic complications.

3. If the divided vagi will regenerate or if permanent impaired gastric motility will result cannot be stated at this time.

4. The operation seems indicated in duodenal and stomal ulcers which do not respond to medical treatment. Gastric ulcers with their probable tendency toward malignant degeneration should be resected.

5. Vagus nerve section may one day have a definite place in the therapy of peptic ulcers, however, the period of observation has been too short for final deductions.

6. The literature pertaining to vagotomy and peptic ulcer has been reviewed.

ADDENDUM

Since this paper was presented 25 additional vagotomies were performed, four of which were transthoracic and 21 transabdominal. The author is still enthusiastic about the procedure.

THE SURGICAL MANAGEMENT OF VARICOSITIES IN THE LOWER EXTREMITY

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IN discussing varicosities of the lower extremity a knowledge of the anatomy, physiology and pathology of the venous system is essential

While the great majority of varicosities occur in the superficial veins, a few do occur in the deeper system.

The longest superficial vein in the body is the great saphenous.[1] It begins in the subcutaneous tissues on the dorsum of the foot, extends upwards in front of the internal malleolus on the anteromedial aspect of the leg, passes posterior to the internal condyle of the knee, up the anteromedial aspect of the thigh to enter the femoral vein at the fossa ovalis. Very important tributaries of this vein join it usually close to its termination. These are important because they frequently become involved in varicosities, and, if not adequately treated may be responsible for recurrence of symptoms. The most important tributary is the external superficial femoral vein which begins in the region of the knee and extends up the anterolateral aspect of the thigh to empty into the long saphenous at or near the fossa ovalis.[2] The external circumflex iliac vein drains the anterolateral aspect of upper part of the thigh and the region of the crest of the ileum. Sometimes it communicates with the external superficial femoral vein. The external pudendal vein drains the area of the scrotum and labia majora and the upper medial portion of the thigh. The superficial epigastric vein drains the lower portion of the abdomen. These tributaries of the long saphenous are of importance because of their possible communications with the long saphenous at lower levels and aiso with the short saphenous vein. Failure of ligation of these tributaries is likely to result in recurrence of the varicosities owing to the retrograde flow into one or both saphenous systems through one or more incompetent tributaries.

The short or external saphenous vein begins on the dorsolateral aspect of the foot, passes upward posterior to the external malleolus and the posterolateral aspect of the leg to enter the popliteal space between the two heads of the gastrocnemius muscle. In the popliteal space it divides into two branches, the larger entering the popliteal vein at its upper end and the other continuing upward posterior to the femur to communicate with the beginning of the profunda femoris vein. Its most important tributary is the femoropopliteal vein which begins in the inferior posterior portion of the thigh and extends inferiorly to the upper part of the popliteal space, where it communicates with that branch of the short saphenous which empties into the profunda femoris. Frequently there is a communication between the long and short saphenous systems

Presented at Annual Meeting Arizona Medical Association, Phoenix, May 19-21, 1948.

in the posterior aspects of the calf of the leg. This must be recognized and corrected.

In addition to the above veins and their tributaries, varicosities may involve veins draining into the internal iliac vein, the most important of which are the superior gluteal, the internal pudendal and the inferior gluteal veins. The superior gluteal vein drains the skin of the buttocks and the gluteal group of muscles passing to the internal iliac vein through the greater sacrosciatic foramen. At times it communicates with varices on the posterior aspect of the thigh. In such instances there may be retrograde flow of blood through them. The internal pudendal originates in the sinuses of the corpus cavernosum and passes posterior through the lesser sacrosciatic foramen to enter the internal iliac vein. Its branches are from the perineum, scrotum and labia majora. Varicosities from them may communicate directly with branches on the inner posterior aspect of the thigh, especially of the external pudendal vein. The inferior gluteal vein accompanies the great sciatic nerve and receives branches from the skin below the lower border of the gluteus maximus muscle. It enters the pelvis through the greater sacrosciatic foramen to enter the internal iliac vein. When varicosed it may communicate with the external or internal saphenous vein.

In addition to the above described superficial veins there are communicating veins that extend between the superficial and deep venous systems. Normally these have little function, but when used the direction of flow is from the superficial to the deep veins through them. In the presence of varicosities they assume great importance because of the possible incompetence of their valves permitting reversal of flow from the deep to the superficial systems.

Normally the flow of blood in the veins of the lower extremity is upward in both the superficial and the deep veins and from the superficial to the deep veins through the communicating veins. The direction of the flow is maintained by the presence of valves in the superficial, communicating, and deep veins. In varicosities as a result of dilatation of the vein and degenerative changes in the valves the latter become incompetent, so that the direction of the flow is reversed, that is from above downward. Such reversal of flow has been demonstrated by the investigations of Brodie, Trendelenburg, Magnus, McPheeters, and Rice.

There is no single cause of varicosities, but there are a number of predisposing and precipitating factors. Of great importance is an inherited predisposition. Varicosities of the lower extremity are frequently associated with varicosities elsewhere in the body, especially those of the hemorrhoidal veins and pampiniform plexus. Vericosities frequently are associated with lesions resulting from connective tissue weakness such as hernias, flat feet and enteroptosis. Also with irritative lesions in the sympathetic ganglia resulting from exogenous factors such as alcohol, tobacco, infections, etc. Endogenous factors as disturbances in metabolism and the endocrine glands, produces venous dilatation and hyperemia. Ultimately inflammation of the vein wall and destruction of the valves occur resulting in varicosities. Their incidence increases with advancing age. Phlebitis and chronic inflammation are frequent contributing causes. Syphilis has received its share of the blame. Antecedent thrombophlebitis of the iliofemoral veins predisposes to varicosities largely because the blocking of the deep system causes a compensatory dilatation of the superficial system which is the only route through which blood from the lower extremity can return. Although infection is a definite factor the number of cases resulting from inflammation is relatively small. Endocrine dysfunction may favor the development of varicosities as evidenced by the rather frequent occurrences of varicosities at those times at which physiologic changes occur, i. e., pregnancy, puberty, and menopause. The importance of pregnancy is shown by the statistics of Oschner and Mahorner. In most cases varicosities appear during the first few weeks of pregnancy before the enlargement of the uterus can possibly have much influence on their formation from pressure. When they occur during these periods of endocrine activity it may be due in part to increased vascularity and stasis in the pelvic veins. Interference with the return flow of venous blood from the lower extremities is an important exciting factor. Obesity, abdominal tumors, such as an enlarged uterus or pelvic neoplasm may produce pressure on pelvic veins, and the presence of the rectum on the left side is thought to be responsible for the slightly greater frequency of left-sided varicosities. Stasis in the lower extremities is especially favored by occupations which require long periods of standing as in clerks, policemen, salespeople and motormen.

The return of venous flow from the lower extremities occurs partly as a result of the pumping action exerted on the deep veins of the extremity by the contraction of the calf muscles during walking. Because of the arrangement of the venous valves the movement of the blood in the deep vein is upward, so that the emptying of the deep veins by muscular contraction permits the flow from the superficial to the deep veins. In the standing position venous statis is, however, favored because of the dependent position of the extremities and loss of the active propulsive power normally exerted on the deep veins by contracting muscles. This vascular stasis in the deep veins interferes with the normal emptying of blood from the superficial veins into them permitting stagnation, dilatation and increased venous pressure.

Varicosities of the superficial veins of the lower extremity consist of dilatation, elongation and tortuosity of the veins. Associated with the dilatation and elongation are degenerative changes and fibrosis which occur in the wall of the vessel resulting in the loss of elasticity. Incompetence of the valves occurs early both as the result of dilatation of the veins and also because of atrophy of the valves. Thrombosis and thrombophlebitis can occur in dilated veins and are usually the result of trauma or infection. Calcification frequently occurs. Varicosities may occur in the communicating collaterals because of the increased venous pressure.

The normal direction of the blood flow is the result of several factors: (a) *vis a tergo*, resulting in flow of blood from the arterial system through the capillary bed into the venous system; (b) pumping action of the muscles of the legs compressing the deep veins forcing the blood upward; (c) the negative intrathoracic pressure. Mitigating against all of these factors is gravity which is considerable in man in the upright position. As a result of the valves in the communicating and the deep veins blood is normally forced from the superficial to the deep system. In varicosities, however, due to the incompetence of these valves the flow is reversed from the deep to the superficial system. Blood may come up through the deep system to the fossa ovalis and regurgitate down the great saphenous, thus resulting in stagnation and partial circulation in the whole lower extremity. This causes an interference of the gaseous, electrolyte and protein exchange between the blood and tissues of the lower extremity. We thus get a lowering of oxygen and an increase of carbon dioxide in the blood. This causes lowered tissue resistance, resulting in early exhaustion, muscle cramps, edema, ulceration and a proneness to infection.

The symptoms in varicose veins are usually mild. Frequently the patient may have no symptoms referable to the vein of which he is conscious because of the insidious onset of the condition. However, after correction of the varicosities the patient becomes aware of the fact that he did have symptoms resulting from the dilated veins because of the marked improvement in well-being following their correction. Because of this insidious onset, many patients complain only of the unsightly venous dilatation. The most frequently encountered symptoms are heaviness, fatigue, burning, itching and rarely a cramp in the extremity. These manifestations are aggravated by standing for long periods of time and are almost invariably relieved by rest and elevation. The dilatations may involve all of both the long and short saphenous veins or only portions or a single branch of either. Dermatitis and later ulcerations are late manifestations.

When examining a patient with varicose veins several tests are used to evaluate the problems presented. In the local examination of the extremity, the examiner should observe skin changes, evidence of venous stasis, the character of the pulse in the dorsalis pedis, posterior tibial, popliteal and femoral arteries.[3] An attempt should be made to elicit Homans' sign. In the presence of an ulcer skin temperature tests are important. The flourescein dye test, where the 5% solution is injected intravenously and the golden green hue is looked for under the illumination of a mercury lamp, is most reliable in testing the circulation of the skin.

The patient stands with the lower limbs exposed to the groin. With one hand of the examiner on the varicose veins while gently percussing it at another level, a fluid wave can be elicited (Schwartz test) when the intervening valves are incompetent. All superficial veins are palpated for tension and pressure within them.

The patient is then placed in a recumbent position, the leg is elevated above the level of the pelvis and the veins are stroked or "milked" toward the groin, thus emptying them. A tourniquet is placed around the upper thigh with sufficient pressure to block the superficial veins.

The patient stands and the veins below the tourniquet may remain collapsed and then fill slowly from below upward in 36 to 60 seconds. The test is repeated, and when the patient stands, the tourniquet is released suddenly. If the veins distend quickly from above downward to their former state of tension, the patient is classified as TRENDELENBURG POSITIVE.

The TRENDELENBURG POSITIVE group comprises those cases in which the valve function in the upper part of the great saphenous vein is deficient or absent, permitting reverse flow from the femoral vein at the saphenofemoral junction. This is the type of case most frequently encountered and it is most effectively treated by preliminary high ligation and division of the great saphenous vein and separate ligation and division of all the tributaries at the fossa ovalis, with subsequent injection therapy. What the tourniquet does in the test is exactly what ligation will do.

The TRENDELENBURG NEGATIVE case is one in which reverse flow from the deep veins into the superficial veins takes place through one or more dilated communicating veins with incompetent valves, sometimes called "blow-outs." In this case the veins fill rapidly from below—less than 35 seconds—when the patient stands while the tourniquet pressure is maintained.

The TRENDELENBURG DOUBLE group comprises those cases in which the back flow takes place at the saphenofemoral junction as well as through one or more of the dilated communicating veins in the lower part of the limb. Briefly it is a combination of the positive and negative types of backflow. The treatment is obviously high ligation and division at the fossa ovalis and ligation and division in the lower part of the limb wherever backflow can be demonstrated.

TRENDELENBURG NIL cases are those with dilated veins in the presence of competent or normal valves. This is the early type of case frequently seen with no reverse flow. These varices may be obliterated by simple sclerosing injections.

While doing these tests the status of the lesser saphenous vein must also be borne in mind. Frequently the reverse flow occurs from the popliteal vein into the lesser saphenous in the popliteal fossa. In these instances the lesser vein must also be ligated.[4]

The PERTHE'S TEST is a simple test to determine patency of the deep veins as well as their functional capacity. A tourniquet is applied above the knee with sufficient pressure to block the superficial veins only, while standing and the veins are distended. The patient is instructed to walk about actively. If, with the tourniquet in place the veins below collapse (empty), then one may assume that there is no serious deep vein blockage and the patient may be treated. On the other hand if the veins become more tense and painful deep venous occlusion must be suspected and conservative treatment given. This test may be performed with a tight elastic bandage applied from the knee to the base of the toes.

In the comparative tourniquet test, the OSCHNER-MAHORNER test, the patient is observed standing, draped so that both legs and thighs are exposed in a good light. The patient walks to and fro in front of the observer. Even with no tourniquet on, the varicosities become less prominent when the patient is walking because the peripheral pumping action of the muscles on the deep veins sucks the blood from the superficial to the deep system. The patient then stops, and a tourniquet is applied around the upper third of the thigh tightly enough to compress the superficial veins and to obstruct the flow of blood through the segment. The patient walks again over the same course at the same speed. The prominence of the varicosities is noted and compared with their prominence when the patient walks without the tourniquet and again when he walks with the tourniquet around the middle third of the thigh and again when he walks with it around the lower third of the thigh. The interpretation is as follows: If a minimum improvement in the varicosities occurs when the tourniquet is at its highest level, and there is no further improvement when it is around the middle third or lower third of the thigh, then only one source exists for retrograde flow, which is through the main opening of the internal saphenous vein into the femoral vein. If there is greater improvement in the varicosities below the tourniquet when it is around the lower third than when it is around the upper third, it is evident that not only is there retrograde flow through the main opening but that there is also a leak through communicating veins below the fossa ovalis. This test combines the advantages of other tests localizing accurately the veins with

incompetent valves. You learn: (1) Whether the valves of the internal saphenous are incompetent; (2) whether the valves of the thigh and leg communicating between the deep and superficial veins are competent; (3) whether the veins communicating between the superficial and deep veins are competent; (4) whether the deep veins are patent.

PRATT TEST.[5] After testing the arterial circulation and performing the Perthe's and Trendelenburg tests one definitely tests for and localizes any incompetent communicating veins. "With the patient lying down, the leg to be tested is elevated and with light massage the veins are emptied. A tourniquet is placed sufficiently high in the thigh to close off the saphenous vein. An Ace bandage is then applied from the toes to the tourniquet. The patient then stands up and the Ace bandage is slowly unwound from above down. With the tourniquet above preventing reflux of femoral blood through the saphenous valves and with the Ace bandage below compressing the remainder of the saphenous vein, a bulge or blowout indicates an incompetent communicating branch vein. Such an area is marked with an indelible pencil and is a point where a secondary ligation will be required. In a limb where there are many such "blowouts" a second Ace bandage is applied from above down. As the first Ace bandage is slowly removed, a blowout appearing between the two Ace bandages is thus a new one and must also be marked and resected. While in most instances there are only one or two such blowouts, occasoinally one finds four or five. Failure to remove all such blowouts results in recurrences."

In the treatment of varicosities in the lower extremity all points to be ligated are marked with an indelible pencil or weak solution of silver nitrate prior to operation in order that the antiseptic solutions used in preparation of the skin will not erase them. The leg and thigh should be draped in a manner to allow the operations performance without having to change the setup.

In nearly all cases high saphenous ligation is imperative. Recurrences are sure to occur unless all of the tributaries at or near the fossa ovalis are clamped, cut and tied. The great saphenous vein must be clamped, cut, and double ligated as near the junction with the femoral as possible, a segment removed and the distal end ligated.

Local anesthesia is preferred to general, because the patient should begin ambulation as soon as he arrives back to his room. When the patient will not tolerate local anesthesia one of the fast acting, rapidly eliminated general anesthetic agents should be used.

The pulsation of the femoral artery is palpated as the vessel passes under the inguinal ligament. A longitudinal incision is made two-fingers' breadth medial to and paralleling the femoral artery. A longitudinal incision facilitates the ligation of all the collaterals. These collaterals are the external pudendal, the circumflex iliac, the superficial epigastric, the thoracoepigastric, and the external superficial femoral. Their relationship to the femoral and external saphenous veins varies considerably. The upper third of the incision is above the fold of the groin, particularly in obese individuals because the fold in the groin is below the inguinal ligament. The incision is extended downward and slightly medialward for about 10 cm. The long saphenous vein lies in the superficial fat and may vary in size. It is isolated by blunt dissection from the tissues over a distance of 3 to 5 cm. from its entrance into the fossa ovalis. The femoral vein lies deep to the deep fascia. In isolating the vein near the fossa ovalis the collaterals come into view. These are doubly clamped, cut and ligated preferably with silk or cotton. The saphenous is divided between clamps as close to the femoral as possible. A distal transfixed ligature is placed, and an ordinary ligature is tied proximal to it. The saphenous vein is then stretched upward from below, clamped, ligated and the portion proximal to the ligature cut away. The vein retracts into the lower end of the wound at this time. The subcutaneous tissues are approximated with fine catgut. The skin is then closed and a pressure dressing is applied.

All blowouts where incompetent communicating veins have been marked should be exposed, the vein ligated above and below it, the communicating vein ligated and the intervening vein removed.

After the "blowout" is resected and the feeding or T-shaped veins divided and tied off, the distal end is transfixed and sutured on tension to the deep fascia.[8] This stretches the tortuous distal end and lifts and suspends the soft spongy tissues. The physical appearance of the ankle is improved at once. The tendency to ulceration is greatly reduced.

After closing and dressing all wounds an Ace

or a pressure bandage should be applied from the base of the toes to the knees, and from above the knee to as near the groin as possible.

The patient should be encouraged to move his legs and walk immediately after surgery, and during convalescence. This facilitates the return to normal function of the deep venous circulation.

It is my custom to have tailormade elastic stockings prepared before surgery. The stockings extend from the base of the toes to below the knee. After healing the patient wears the stockings during ambulatory hours for three to six weeks. This keeps the venous segments between the points of ligation and their radicles collapsed at all times. Surprisingly enough many of these cases never need subsequent injections with sclerosing agents when this is done.

Of the dangers encountered in ligations embolism probably heads the list. By careful technique and early ambulation the tendency for venous thrombosis and embolism is lessened. In those patients showing an early Homans' sign or any other suspicion of thrombosis, heparin followed with dicoumerol with proper checking of clotting time and prothrombin time should be instituted at once.

There are reports of the mistaken ligation of the femoral artery and the femoral vein. This can be avoided if all bleeding points are caught and immediately ligated as the operation progresses. The leaving of clamps on these bleeders until the last part of the operation is not only sloppy technique, but frequently the femoral artery itself may be squeezed in the tissues by a nearby clamp sufficient to obliterate pulsation in the artery, permitting the operator to ligate and excise a piece of it before the error is recognized. The femoral vein always lies beneath the deep fascia. By keeping the operative field dry, using blunt dissection in the superficial fat, and not ligating large veins until good exposure is obtained, injury to it can be avoided.

An uncommon postoperative complication is lymphorrhea. It is almost impossible to avoid the large or small lymphatic trunks.[6] These are either cut or torn by vigorous retraction. A fluctuant tumor usually appears beneath the incision a few days after operation. When drained a clear straw-colored fluid is evacuated. This drainage may persist, or reaccumulate for weeks or months. This is usually controlled by bed rest, elevation of the extremity and application of elastic pressure from the base of the toes to above the operation site. Sometimes packing the wound with a thromboplastic material plus the above treatment is necessary.

Wound infection is dreaded and avoided if possible by sterile technique. If it occurs the stitches should be removed, heat applied and the antibiotics administered.

Injecting the veins with sclerosing solutions has been very popular. This method of treatment is losing its popularity because of the increasing number of recanalization, deep venous thrombosis, pain and allergic reactions.

There are numerous sclerosing agents used, the most popular being sodium morrhuate. Others are sodium recinoleate, sodium psylliate, monoethanolamine oleate, sodium linoleate, jecoroleate, potassium oleate, or the soaps. To the group acting by osmosis belong the crystalloids such as: sodium chloride, calcium chloride, sodium citrate, dextrose, saccharose, invert sugar, and combinations of these. The heavy metals are also used, such as: biehloride of mercury, mercuric iodide, and iron perchloride. Certain organic compounds such as: sodium salicylate, quinine hydrochloride, and urethane are used. The halogens have been resorted to, as: tincture of iodine, Lugol's solution and Pregl's solution.

As stated above, injections are indicated in those cases showing the Trendelenburg nil test. In these the valves are competent but the veins are dilated. Injections are also used in conjunction with and several days or weeks after surgery. Retrograde injection should never be used at the time of surgery.[7] The disasters are usually due to deep venous thrombosis due to the entry of the sclerosing solution into the deep venous group through communicating veins.[9] High ligation of the saphenous vein with multiple ligations at the "bursts" has given uniformly the best results with fewer complications.[10] In some cases the Mayo vein stripper can be used to advantage, but by careful evaluation beforehand, a much neater result is obtained by the above method.

It is wise to test the sensitivity of the patient to the sclerosing solution before actually injecting the veins. This is done by injecting about ¼ c.c. of the solution into the vein a day prior to the actual treatment. If no untoward events are noticed, the solution is considered safe.

One to two cubic centimeters are injected into each varicosity, never using over 5 c.c. in mul-

tiple injections at one sitting. Some operators have the patient stand and after inserting the needle inject the vein. Another method is to have the patient lie down and insert the needle into the vein. By exerting pressure on the vein with the thumb and forefinger of the free hand above and below the site of injection, the solution is injected and held in the segment while it is evenly massaged throughout the segment with the other hand.

The writer prefers to use a tourniquet above and below the site of injection. A tourniquet is placed above the site, the needle is inserted and an assistant then places a tourniquet below. The limb is then elevated, and the proximal tourniquet released. As soon as all the blood has drained out the proximal tourniquet is replaced by the assistant. This leaves the needle in the collapsed segment between the tourniquets. One to two cubic centimeters of the sclerosing solution is then injected. The venous segment is massaged thoroughly so as to get good distribution of the solution. An Ace bandage is then placed from the base of the toes to and above the tourniquet, releasing each as it is approached. This method keeps the irritated intima collapsed and gives better assurance of more permanent obliteration.

Regardless of whether ligation and section, multiple ligations and sections alone, or injections alone or in combination are used, a most careful follow-up must be instituted or recurrence will occur. The patients must have elastic bandages or stockings on immediately after treatment. Tailor-made elastic stockings are preferred. The elastic binders must be worn all the time when the patient is ambulatory for a minimum of three weeks postoperatively. He should be ambulatory all the time to discourage deep vein thrombosis, and to encourage the normal resumption of the deep venous circulation.

A paper of this type is not complete without dealing with varicose ulceration, skin infections and phlebitis.

Injection therapy is contraindicated in the presence of infection. Ligation at the upper end of the vein is sometimes indicated in the presence of an ascending infection and thmrombosis. The antibiotics are indicated along with rest, local heat, compression and local antiseptics when the causative organism can be ascertained.

Ulceration is usually initiated by trauma on an area of skin, frequently just above the ankle, where stasis has devitalized the tissue. Frequently it is complicated by a fungus infection. This may be cleared up with permanganate of potash solution soaks, painting with tincture gentian violet and/or the use of some form of Whitfield's ointment. All efforts should be exerted toward obtaining a better blood supply to the area. The patient should sleep with the foot of his bed elevated at least six inches to encourage venous return from the extremity. A sponge rubber pad should be placed over the area and held in place by an elastic bandage extending from the base of the toes to the popliteal space while ambulatory. If a tailor-made elastic stocking is used a zipper can be sewed into it to facilitate the placing of the sponge and dressings in the proper location.[11] In recent years chlorophyll has been found to be of great help locally on indolent ulcers. It seems to stimulate healing and keeps down the odor. One may get the same results by the use of Unna's boot.

In varicose ulcers the underlying cause is the varix that feeds them. Therefore, it is felt that as soon as infection in the ulcer is under control, ligation and section of all varicosities should be done. This means that the greater saphenous vein and all branches found near the fossa ovalis must be separately ligated, and, that all "blowouts" in the thigh or leg must be ligated and sectioned.

Usually following such ligation and subsequent injection of any remaining varicosities with sclerosing solution, the ulcer shows marked improvement. If, however, the ulcer persists, further investigation of the area about the ulcer should be made. Here skin temperatures and fluorescein dye tests are a great help. Frequently the ulcer may be situated over a pool of varicosities into which an incompetent perforating branch from the deep system is feeding deoxygenated blood. In some cases scar tissue surrounds the ulcer and interferes with the blood supply. Most often the cause of non-healing of the ulcer in the face of adequate treatment of the associated varicosities will be found in the poor type of epithelial tissue being formed from the ulcer margins.

In cases of arteriosclerosis and venous stagnation physiotherapy benefits. In the presence of "blowouts," dense scar tissue and poor epithelial tissues plastic operations are indicated. A wide elliptical incision is made, outside the ring of scar tissue and down to the fascia. The

intervening tissue is removed "en bloc." All varicosities are ligated. The resulting wound is allowed to granulate in. At a later date the defect is covered with pinch grafts (Reverdin type). These should be covered with a thrombin topical solution, covered with a paraffin mesh gauze and a pressure dressing applied. On or about the tenth day the dressings are removed. Usually one gets a very high percentage of takes.

Pinch grafts are more successful than sliding or large sheet grafts, because they seem to establish better circulation.

SUMMARY

1. As a basis for the surgical management of varicosities in the lower extremity one should be thoroughly acquainted with: (a) the anatomy of the superficial and deep venous systems; (b) the normal and pathological physiology of the circulation of the extremity; (c) the etiological factors and (d) the pathology involved.

2. In examining the patient one should understand the principles and interpretations of all the tests such as: TRENDELENBURG POSITIVE, TRENDELENBURG NEGATIVE, TRENDELENBURG DOUBLE, and TRENDELENBURG NIL; The PERTHE'S TEST: The comparative tourniquet tests of OSCHNER-MAHORNER, and the PRATT elastic bandage test.

3. Excellent surgical technique is a prerequisite.

4. A prolonged and most careful follow-up is essential to prevent recurrences and complication.

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ACUTE OBSTRUCTIVE LARYNGOTRACHEOBRONCHITIS:* Management from the Standpoint of the General Practitioner and Pediatrician

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ACUTE obstructive laryngotracheobronchitis is a very serious, but luckily a relatively uncommon disease. There are many who have never had such a case in their private practice. Some, however, have been less fortunate and the experience may have left you baffled and in dread of being called in to take care of another child who suffers from this terrible illness. To dispel some of this fear I shall try to present a comprehensive and practical picture of the management of these cases—from the standpoint of the general practitioner and pediatrician.

When called in to see a child in acute respiratory distress, four questions will naturally arise. First, "Is this a case of croup?" that is, "Is this respiratory distress due to an acute infectious obstruction of the laryngotracheal airway?" Second, "If it is, then what is the etiology of the infection?" Third, "Is it safe for me to keep this child at home and observe it under palliative treatment or must I send it to the hospital at once for possible emergency treatment?" And fourth, "If I decide to keep it at home, how am I going to treat it?" I will try to answer these four questions as fully as space will permit and then I will discuss briefly a few serious complications that can occur after tracheotomy and which belong to the domain of the general practitioner and pediatrician as well as the otolaryngologist and which unfortunately are often overlooked by both.

The first question—Is the respiratory distress of the child due to an acute obstruction of the laryngotracheal airway? Almost all conditions that cause respiraory difficulty or irregularity have been sent into the Willard Parker Hospital wrongly diagnosed as croup, usually as diph-

* Presented before Arizona Medical Association, annual meeting May 19-21, 1948, Phoenix.

theritic croup. I will mention only a few of the more common conditions thus misdiagnosed, such as: tonsillitis, peritonsillar abscess, and retropharyngeal abscess with some respiratory difficulty or only voice changes; anterior poliomyelitis with intercostal or diaphragmatic paralysis with some weakness of the voice; convulsions from tetany, epilepsy, meningitis, pertussis, with temporary respiratory irregularity and spasms; diabetic coma; pneumonia, pertussis, asthma, and heart failure with attendant respiratory distress.

To confirm the diagnosis of acute laryngotracheal obstruction one must, first, establish the presence of a mechanical block in the airway. This is shown by stridor, retractions of the mobile parts of the chest wall during inspiration, ballooning of the neck and contraction of the abdominal muscles during expiration, and suppression of the breath sounds. Retractions are due to the unequal expansion of the chest wall and the lungs. Because of the laryngeal obstruction the lungs do not fill rapidly enough and this lag results in an increased negative pleural pressure producing an indrawing of the mobile parts of the chest wall. The retractions appear first in the suprasternal and supraclavicular regions, later in the xiphoid region and the intercostal spaces and in cases of very marked obstructions the entire sternum seems to touch the vertebral column. The deeper the retractions the more severe is the obstruction. However, younger patients have a more mobile chest wall and the retractions will be deeper with a given degree of stenosis. In adults, even the most severe obstructions do not cause retractions of the chest wall because it is already fixed; all that is seen is suprasternal and supraclavicular retractions and overworking of the accessory muscles of respiration. On the other hand, in young infants the chest wall is so extremely mobile that any respiratory strain such as occurs in pneumonia, asthma or heart failure, will result in mild or moderate retractions. Do not be misled into thinking that such an infant has laryngotracheal obstruction. One more word of caution: When examining the patient be sure that the difficulty is really *inspiratory* and not expiratory. Time and again I have been called in to see infants under one year of age diagnosed as having severe "croups" and I have found that the difficulty was entirely expiratory. The cause was evidently bronchial in origin—usually bronchial asthma. The obstruction can be so severe that no rales are heard If adrenalin is administered, piping rales will usually appear after a few minutes and the diagnosis of asthma will be confirmed.

When it is decided that the child has an obstruction in the laryngotracheal airway, the next question is: What is the etiology? In a paper on Acute Infectious Croup[1] in 1934, Dr. Wishik and I listed over 70 possible causes of acute laryngotracheal obstruction, and today several more can be added. It is impossible for me to discuss all of these conditions. Briefly, what we generally have to determine is whether the obstruction is due to diphtheria, whether it is associated with one of the contagious diseases (scarlet fever, measles, pertussis, and varicella); whether it is due to a non-specific infection; or possibly to aspiration of a foreign body.

The parents should be carefully questioned regarding the result and the date of the Schick test; possible exposure to contagious diseases; history of past contagious diseases; allergy in the child or family, and the possibility, even though it be remote, of aspiration of a foreign body. Also inquire whether the child was hoarse previous to the present illness.

A most important fact to determine is the presence of hoarseness. If the child is old enough to talk, ask him a question and note whether he is hoarse. If he is too young to talk, make him cry and note whether there is any hoarseness. Do not mistake weakness of the voice—as can occur in anterior poliomyelitis—or "hot-potato" speech as in peritonsillar abscess, for hoarseness. If the child is hoarse the respiratory obstruction is associated with a laryngitis and the chances are good that you are dealing with a case of acute infectious croup. If the voice is clear, it is still possible that the patient has an acute infectious obstruction of the tracheobronchial airway without vocal cord involvement. However, if the voice is clear you should look for some other cause of the obstruction.

Let us assume that you are dealing with acute obstructive laryngotracheobronchitis. The next thing is to rule out diphtheria. Unless you are absolutely sure that the patient has had a reliably negative Schick test within the past six months, diphtheria must be considered until it is proven otherwise. Certain points will help make a tentative diagnosis without recourse to direct laryngoscopy. However, before going into that, I would like to discuss briefly the living

pathology of acute obstructive laryngotracheobronchitis because it will help to better understand the symptomatology of this disease.

In both the diphtheritic and the non-specific infections the obstruction is due to the *same* four factors: namely, edema, membrane formation, mucopurulent exudation, and spasm with fixation of the vocal cords. In both types of infection these factors of obstruction vary only in frequency and in degree. In diphtheria the prime factor is usually the membrane formation, whereas in the non-specific infections the obstruction is usually due to edema, with or without mucopurulent secretion. However, we have seen early cases of diphtheria in which the obstruction was due entirely to edema, and in which the membrane developed a day or two after the patient was admitted to the hospital. We have also seen cases of streptococcus, staphylococcus or pneumococcus laryngitis in which the obstruction was due mainly to thick fibrinous membranes which were indistinguishable, grossly, from diphtheritic membranes. With these facts in mind, the symptomatology should be easier to understand.

The onset in diphtheria is more gradual—with malaise, sore throat, rhinitis, low-grade fever and cough, followed by hoarseness and later dyspnea. (Since the obstruction is generally due to a membrane, cough and hoarseness appear first, to be followed by dyspnea when the membrane becomes thick enough to cause obstruction.) On the other hand, in the non-specific infections, edema is usually the predominant factor at first and the onset is as a rule much more sudden. There may be no preceding sore throat and the dyspnea is usually out of all proportion to the slight hoarseness. (Since there is very little submucous areolar tissue over the true vocal cords, swelling of the vocal cords cannot become marked and the hoarseness is usually not so striking.) If a child is seen who has severe retractions and only slight hoarseness the chances are that the patient does not have diphtheria. On the other hand, a child who has mild retractions and complete aphonia, should be strongly suspected of having diphtheria. Of course, this is not an absolute rule, but it will be true in the greater majority of instances. Further, in diphtheritic laryngotracheitis the dyspnea is progressive, usually without the remissions which are characteristic of the non-specific infections. However, remissions can occur if the child coughs up some of the membrane, which it swallows unbeknown to the parents. You should also bear in mind the occasional occurrence of tracheobronchial diphtheria in which there is no hoarseness, and which is usually misdiagnosed as pneumonia or asthma.

Of course, the nose and throat should be carefully examined for the presence of a membrane—the nose in particular, because it is often overlooked there.

From the history and the physical examination a tentative diagnosis can be established, but to be certain a direct laryngoscopy and bacteriological examination is essential. As someone once said, stridor should not only be heard, but also seen. When possible, any child who has more than mild signs of inspiratory obstruction should be examined by direct laryngoscopy. This procedure is harmless in expert hands and yields a wealth of information of value in making a diagnosis, and in treatment. In occasional cases even a direct laryngoscopic examination will not give conclusive information; therefore, in all cases direct cultures from the larynx should be made. We have checked the reliability of a single direct laryngeal culture and found 20 per cent negative reports in positive diphtheria cases. By using two laboratories (Willard Parker Hospital and the New York City Board of Health) this error was reduced to 11 per cent. Therefore, cultures from the nose and throat should be taken at the same time.

When confronted with a case of acute obstructive non-specific laryngotracheobronchitis, it must be decided whether it is safe to observe and treat the child at his home.

Almost everyone has had experience with the type of case in which the child awakens in the middle of the night with a sudden loud stridor and retractions, and the condition appears quite alarming. You gave the child an expectorant and a sedative, the stridor soon cleared, and the next night the same scene was re-enacted. This is the typical case of spasmodic croup, due to a spasm of the vocal cords, which is so alarming and yet relatively devoid of danger.

The type of obstructive laryngitis which I wish to discuss has an onset which is usually, although not always, less dramatic, but the child shows signs of progressive obstruction. The retractions grow deeper, the skin of the neck balloons out with each expiration, the upper abdominal muscles show strong expulsive

contractions, the breath sounds become suppressed throughout the chest, and the child becomes restless. It wakes up every few minutes and begins to have an anxious look. If the obstruction progresses over a long period of time, he becomes ashen pale, with bouts of cyanosis due to temporary asphyxial attacks. He becomes more and more restless, turning from side to side, getting up and lying down, with a pleading look, dead tired, but unable to rest. If unrelieved the child becomes exhausted; the pulse becomes very rapid and feeble, the respirations become rapid and shallow, the retractions become less marked or disappear entirely, because he is too weak to produce retractions, the temperature climbs to 105°, 107°, or 108°F., and soon stupor, coma, convulsions and death follow.

What has just been described is the progression of events that usually takes place in a case of unrelieved acute laryngotracheobronchitis, in which the obstruction occurs subglottically, i.e., below the vocal cords. This manner of development gives the doctor a good margin of safety and permits him to wait for improvement before resorting to operative relief of the obstruction. However, there is one type of acute laryngeal obstruction which is one of the most treacherous to be found in the practice of medicine. It occurs in 10 per cent to 20 per cent of all cases, depending upon the epidemic, and is due to edema of the supraglottic structures of the larynx—namely, the epiglottis, aryepiglottic folds and arytenoids. The general appearance of these patients often belies the gravity of their condition. Even when there is tremendous edema and impending asphyxia, the patient may be quite comfortable, of good color, and may have only mild or moderate suprasternal and supraclavicular retractions. Because of this lack of distress the doctor is naturally led to feel that he can safely observe this patient just as he has other patients with "mild croup." That is where the danger lies! With catastrophic suddenness the patient's condition may be radically altered. A fit of coughing or even just a change in the position of the body may so displace the waterlogged, flabby, supraglottic structures that they are suddenly pulled into the glottis. With each succeeding, frantic, inspiratory effort the tissues are jammed down more tightly and the patient may become asphyxiated in a few moments under your very eyes. In 1932 I originally noted and later described[2] the characteristic respiratory sounds heard in patients with supraglottic edema. There is a low-pitched inspiratory stertor followed by a louder and lower-pitched, coarse, expiratory rattle, resembling a snore. By performing the act of gurgling, with the mouth dry, and allowing the relaxed tissues of the throat to vibrate an idea of this coarse expiratory rattle can be obtained. If there is extensive edema present, the inspiratory stertor disappears, but the expiratory rattle always remains. Should this sound be heard, the patient being awake, of course, a confirmatory laryngoscopy should be done at once, a bronchoscope passed, and a tracheotomy performed immediately.

The decision as to whether it is safe to observe and treat the child at his home will necessarily depend upon the severity of the obstruction, the duration of the obstruction, and whether or not the child is getting worse. The type of obstruction—i.e., whether it is subglottic and, therefore, less apt to become suddenly worse, or supraglottic, which may cause sudden asphyxiation—is, of course, a very important factor. Above all, the previous experience that the physician in charge has had with this type of case will generally be a determining factor as to which child he can safely keep at home. From my observations I would say, for the average pediatrician, any case of more than moderate severity should be sent to an adequately equipped hospital where it can be watched and given expert emergency treatment if necessary. However, if you are going to observe this type of case at home let me warn you to keep in close touch with the child yourself. See it yourself every hour or two. If possible, get a special nurse—with intelligence—and explain to her in detail what she should be looking for. Do not let the parents be the judge of the child's condition and wait for them to call you. Because of reliance on the judgment of the parents, children are often admitted to the hospital in extremis.

The last question is, "What is the treatment from the standpoint of the general practitioner and the pediatrician?" This can be divided into (1) General and (2) Specific Treatment.

The general treatment is very important. I have seen many patients admitted to the hospital who, on admission, seemed to be in urgent need of operative intervention, but who were tided over by palliative treatment, and who made undramatic recoveries. Except in extreme cases,

one should not observe a patient for only five or ten minutes before deciding to perform an operation. We have observed patients for twelve to twenty-four hours while employing various methods to tide them over this crucial period, thus avoiding many operations and post-operative complications.

The general treatment is as follows:

(1) Rest: This is most important. It means avoiding needless examinations and extra manipulations by special nurses who feel they have to earn their salary. Do not disturb the sleeping child for anything, least of all for medication which is only presumed to be of value. Group all treatments and manipulations during one period and allow the child undisturbed sleep.

(2) Sedation: Opiates or atropine should not be used. The opiates cut down the beneficial cough reflex and may inhibit restlessness, which is such a valuable index of air hunger. The atropine causes a drying of laryngotracheobronchial secretions so that they become gummy and inspissated and act as a foreign body causing obstruction. For sedation we have used whiskey diluted with water and sugar, small doses of bromides or phenobarbital.

(3) As an expectorant, syrup of ipecac can be given in doses of ten minims to a dram, depending on the age, every three hours; if this induces vomiting, stop the ipecac.

(4) Hyperpyrexia: For temperatures over 102°F. various anti-pyretic measures should be taken, depending upon the degree of the fever; e.g. continuous sponges, wet packs, ice caps, or cold colonic irrigations. Aspirin can be given. Lowering of the fever results in a lowering of the metabolic rate, therefore lessening the oxygen requirements so that the respiratory distress is diminished. I have seen patients who seemed to require urgent operative intervention and, after lowering the temperature two to four degrees, they took a quick turn for the better. This is a very important detail in the treatment.

(5) Fluids: I do not need to discuss the importance of fluids. However, I would like to caution you that giving a child a bottle to pull on merely adds to the respiratory strain of an already overburdened baby. Use a Breck feeder or a catheter-tipped medicine dropper and squirt the fluid into the side of the mouth a little at a time. If fluids are refused by mouth, they should be given parenterally, but be careful not to overburden the heart.

(6) Steam: This is the traditional and orthodox treatment for croup and in most cases it is highly effective. If given, be sure the tent or cubicle is almost saturated with moisture. Many times a croup kettle merely heats the surrounding air and the relative humidity is actually lowered. The use of a humidifier is to be recommended, at a temperature of 70°F. and a relative humidity of 80 to 90. If it is too warm it is depressing both to the patient and the nurse. In some cases, steam is definitely harmful. I have seen children, particularly those in whom the obstruction was due to subglottic edema with very little mucopurulent secretion, who did poorly under a steam tent, but on changing to an oxygen tent or just cool air at about 60° to 65°F., started to improve rapidly. One must always remember that children react differently and various methods must be tried in order to get the best results. The use of helium and oxygen, under pressure, has also been tried with good results.

Specific Treatment: For all practical purposes this means the use of chemotherapy and the antibiotics, although occasionally serotherapy is used, e. g. scarlet fever antitoxin, pneumonia serum and influenza antitoxin. The scope of this paper is too limited to permit me to go into detailed statistics regarding the results of the use of sulfa drugs and the antibiotics. Suffice it to say that although the complications and the mortality rate have been decreased, the need for operative intervention seems to have been undiminishd by these drugs. This can be accounted for by the fact that this disease is primarily due to a virus and the complications are due to secondary invaders. I do not have the time here to go into the details of the reasons for this view. During a discussion of the 1938-1940 epidemic of this disease before the New York Academy of Medicine[5] I remarked that there were certain similarities between laryngotracheobronchitis complicating measles and laryngotracheobronchitis occurring during this epidemic—namely the high incidence of Staph. aureus, the tendency to ulceration, and the leukopenia; which prompted me to believe that we were dealing with a virus infection. Since then, this concept seems to have become the general consensus of opinion.

A word about the operative treatment: There are three procedures at our disposal—namely, aspiration, intubation and tracheotomy. In most

cases the first will suffice. It is simple and effective when the correct indications are present, namely, mucopurulent secretions. Intubation is indicated when the obstruction is due to subglottic edema. It should always be used together with aspiration—and only in the hands of experienced men. Furthermore, when it is done someone MUST be present *at all times* to replace the tube at once if it becomes blocked-up or is coughed up. In recent years the pendulum has swung to the use of a tracheotomy. The operation is a simple one, but the post-operative care has to be very good if dangerous complications are to be avoided. I will not discuss the rationale, indications and contra-indications, the dangers and pitfalls of these procedures. If there are any questions I shall be glad to try to answer them. Those interested in a detailed study are referred to several of my papers.[1, 3, 6]

I would like to discuss briefly a few serious complications which can occur after tracheotomy and which are of interest to the general practitioner or pediatrician as well as to the otolaryngologist.

(1) *Hyperpyrexia:* In 16 cases (13 per cent of the 126 tracheotomies which were performed during a recent ten-year period at the Willard Parker Hospital) the temperatures rose to 105°F. - 108°F. following operation. This complication must be watched for by taking the temperatures every hour routinely, after the operation. during the first four to six hours. Vigorous anti-pyretic measures should be started at once and not stopped until the fever is diminished.

(2) *Extreme restlessness and convulsions* occurred in 9 cases in this series.[3] The causes were cerebral hemorrhages after prolonged anoxia, alkalosis, hyperpyrexia, air hunger, beginning meningitis, tension pneumothorax and chemotherapy. These possibilities must be borne in mind and appropriate measures taken to combat them.

(3) *Dangers from Oxygen:* There is a general tendency to give oxygen whenever there is any respiratory distress. Most of the time it is good, but after tracheotomy the use of oxygen is dangerous because of its dessicating effect on the tracheobronchial secretions. Once these secretions become crusted they act as foreign bodies and produce the most serious complications. Do not order oxygen unless the child has pneumonia and cyanosis. Moreover, if it is used it should be combined with 5 per cent to 10 per cent carbondioxide and steam, together with instillations of warm dilute sodium bicarbonate solution (5 per cent) into the trachea to combat the drying effect of the oxygen.

(4) *Acidosis and Alkalosis:* Patients who have prolonged respiratory obstruction tend to develop acidosis due to the accumulation of carbondioxide and the acids formed because of overwork; also because of the diminished intake of bases and fluids. Particularly in very young children, the acid-base balance is easily upset and must be watched carefully. A much less common occurrence, but more serious because it is often overlooked, is the development of alkalosis. After tracheotomy for prolonged respiratory obstruction, several patients continued to breathe rapidly and deeply for long periods. This hyperventilation with the blowing-off of large amounts of carbon dioxide resulted in an alkalosis accompanied by the usual signs of tetany—such as carpopedal spasm, twitchings and convulsions. These patients should be given carbon dioxide-oxygen mixture to breathe, saline solution by vein, and calcium gluconate intramuscularly or intravenously. The carbon dioxide concentration and carbon dioxide combining power of the blood should be followed carefully.

(5) *Tension Pneumothorax.* The scope of this paper does not permit more than a few remarks. Those interested are referred to a discussion by the author.[4] Most of the otolaryngologists and pediatricians are not keenly aware of the occurrence of tension pneumothorax and are not on the lookout for its occurrence. Moreover, unless the post mortem examination is done under water, or x-rays are taken after death, its presence will be missed. There were 17 cases of tension pneumothorax, an incidence of 13.5 per cent, in the series of 126 tracheotomies for non-specific obstructive laryngotracheobronchitis at the Willard Parker Hospital during a recent decade. Of these eight were unilateral and nine were bilateral. The mortality rate in cases of unilateral pneumothorax is about 20 per cent, and in the bilateral type about 90 per cent; 60 per cent of these pneumothoracies occurred within a few hours after the operation. However, it can occur before, during, immediately after and as late as three days after an operation.

Prophylaxis is the concern of the otolaryngologist, but the diagnosis concerns both the general practitioner and the pediatrician. The symptoms

depend upon the rapidity of the onset, the size of the pneumothorax and whether it is unilateral or bilateral. Thus, patients may be almost symptom-free or they may go into shock, become cyanotic and stop breathing.

The physical signs are:

(1) Dyspnea with diminished volume and force of the air blast coming from the tracheotomy tube (after making certain that the airway is clear).

(2) Diminished excursion of the affected side with displacement of the heart to the opposite side.

(3) Diminished breath and voice sounds with hyperresonance or tympany.

This condition may be mistaken for pulmonary emphysema and therefore all patients should have a roentgenogram made routinely before and as soon after tracheotomy as is feasible, and as often thereafter as seems warranted.

Treatment: In cases where a small pneumothorax is present which causes no embarrassment, no treatment is needed. Only careful observation is necessary. It will usually be absorbed within a few days. In a tension pneumothorax—in which the pressure is above the atmospheric pressure—immediate and continuous decompression is necessary. This should be done slowly and with a minimum of manipulation in order to avoid a too rapid shift of the mediastinum, and consequent shock. One of the best methods of decompression is by means of a closed underwater drainage (similar to that used for the treatment of empyema of the chest). A large dull needle is inserted into the affected pleural cavity and is connected with a rubber tube, the distal end of which is placed in some antiseptic solution such as 1:10,000 potassium permanganate, about 18 inches below the chest. If a piece of glass tube is interposed within the rubber tube the fluid will be seen to rise and fluctuate freely when all the free air has been removed. At this point the tube may be clamped and the patient watched. When distress is absent the needle may be withdrawn from the chest. Another method is to attach a finger cot, with the tip cut off, to the hub of the needle in the chest. This will act as a one-way flapper valve, allowing air to get out, but not to come in. A third method is to use a pneumothorax apparatus, reversing it and removing the air slowly until the pressure is minus 6 centimeters to minus 10 centimeters of water, and repeat this as often as is needed.

It must be remembered that early recognition and immediate and continued treatment of this condition are life-saving. The possibility of the occurrence of tension pneumothorax must always be borne in mind and watched for after a tracheotomy.

In conclusion I would like to say this: The treatment of these cases is so specialized, the prognosis so serious, and the opportunity for acquiring intimate knowledge through personal experience is usually so small that I feel it would be wise for county hospitals to assign the care of these patients to a small group of general practitioners, pediatricians and laryngologists who could thus cooperate and become more familiar with the special problems encountered in this disease.

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Arizona Medical Problems

CONSULTATION AND CASE ANALYSIS

ARIZONA MEDICINE again presents an unsolved and difficult case from the practice of Arizona physicians, with the Case-Analysis and comments of a specially-chosen and nationally-known Consultant.

Any physician who has an undiagnosed case which has defied other methods of solution may send it for consideration. The case should be completely worked up, but an editor will help compose the report. Whenever the need for an answer is urgent, the Consultant's reply will be sent direct to the submitting physician, before publication.

Please send communications and data to Dr. W. H. Oatway, Jr., 123 S. Stone Avenue, Tucson, Arizona, or care of The Editor, Arizona Medicine.

The CONSULTANT in this case is *Dr. John H. Urabec* of Los Angeles, a well-known California specialist in diseases of the chest.

Dr. Urabec was trained at the University of Pennsylvania Medical School, at Barlow Sanatorium in Los Angeles, at Belleview Hospital in New York as a resident physician, and at the Los Angeles County Hospital as head physician. He served in the Medical Corps, USA., as a major. He is instructor in medicine at the University of Southern California Medical School; is on the attendant staffs of the Good Samaritan Hospital, Barlow Sanatorium, and the Veterans Administration Hospital; and is attending physician for bronchoscopy at the Los Angeles County Hospital.

Dr. Urabec is a member of several societies, including the American and California Trudeau Societies, and is now chairman of the X-ray survey committee for Los Angeles County.

CASE NUMBER XII

The patient is **a white girl, 8 years of age.** She was cared for by a pediatrician, and seen later by a specialist in diseases of the chest. The child is a native of New York, but has been living in Tucson with her family for the past two years. Her only illnesses have been measles and chickenpox, and her tonsils were removed four years ago. Her mother has been well, but her father died of bronchiectasis a year ago. They had no other children.

The **chief complaint** at the time of consultation was the persistence of a respiratory illness of six weeks duration.

She had been well until May, when she had **an acute grippe-like** infection, with a fever of 100 to 101 degrees for several days, and evidences of an upper and lower respiratory infection. The severity of the illness waned, but the slightly productive cough continued; she had an afternoon fever of about 99.6 degrees, and she was underweight, listless and irritable.

This condition persisted for several weeks, and x-rays of the sinuses and chest were then obtained. The sinuses were clear, but **in the chest film** there was a patch of hazy infiltration, about 5 x 6 cm. in size, just above the dome of the right diaphragm. It did not involve the lateral sulcus, but the trunks to the hilum were accentuated, and there was a probable enlargement of the hilar nodes on that side.

She was taken to a hospital **for further study.** The blood count showed a mild secondary anemia, a leucocytosis of 10,250 cells, with 68% PMN, 24% lymphocytes, 4% monocytes, and 4% eosinophiles. A urinalysis was normal. Two scanty sputum specimens were negative by concentrate smear for tubercle bacilli and fungi. She was given 2 grams of sulfadiazine a day for 4 days without effect on the fever or symptoms. She was allowed to go home, but a consultation was requested by the physician.

At the time of consultation the history was found to be essentially as reported. The throat had been slightly sore, but there were no ear symptoms, there was no chest pain, and no wheezing had been present since the acute illness.

The child was thin and slightly pallid. The nose was normal, the throat was not inflamed, and the tonsils had been cleanly removed. The lymph nodes were normal in size, the heart was normal, and the liver and spleen were not palpable. The special senses, reflexes, bones and joints, and skin were normal. The lungs were normal except for an area of impaired percussion off and below the hilum, anteriorly on the right. In the same area there were accentuated breath sounds, but no rales. Fluoroscopy showed the lesion seen by x-ray, except that it was triangular and retracted slightly toward the hilum. The diaphragm was slightly elevated and restricted in mobility.

Another x-ray of the chest was suggested, and this confirmed the change seen in fluoroscopy. The lesion was smaller (5 x 5 x 5 cm.) but slightly more dense. Skin tests were suggested, and there was a 2 plus reaction to the first strength of PPD tuberculin, and a 3 plus reaction to 1:100 coccidioidin. A gastric aspiration was done, after another sputum had been found negative; the specimen contained some purulent material, but the culture and animal inoculation were negative for tubercle bacilli and fungi.

The problem of diagnosis and prognosis became obscure at this point.

QUESTIONS:

1. What is the probable diagnosis?
2. What further diagnostic measures could or should be used?
3. How should the case be treated, considering the various possibilities?
4. What is the prognosis?

M. D., Tucson.

CASE ANALYSIS AND ANSWERS—

A definite *diagnosis* cannot be established with certainty from the information available above.

It is a difficult problem. The laboratory studies are equivocal. The roentgenological findings are consistent with *either a primary tuberculosis* or *a coccidioidomycosis.*

The initial lesion appears in the lower portion of the lung field which is the most common site of these two infections. The presence of both positive tuberculin and coccidioidin tests adds to the difficulty in the problem. The degree of reaction of the skin tests is of no help, since usually in primary infections of tuberculosis and coccidioidomycosis without complications the skin sensitivity is increased. Furthermore both infections follow the Law of Parrot, in which there is a bipolar infection, that is, a parenchymal infection as well as an infection of the draining lymph nodes. Because the patient lived in an endemic area for coccidioidomycosis, this infection certainly must be ruled out.

Primary infection with coccidioides immitis in a child is usually a mild infection; consequently *the initial impression* of this case would be that of a *primary pulmonary tuberculosis* with associated infection and enlargement of the tracheobronchial glands followed by compression and atalectasis of the right middle lobe. Instances of such have been observed at the Children's Hospital in Los Angeles where atelectasis of one or more lobes has occurred from the compression caused by tuberculous tracheobronchial lymph nodes.

The most important of the diagnostic measures which would aid in determining the nature of the disease process present probably would be *the complement fixation and precipitin tests* for coccidioidomycosis, principally to rule out or establish this diagnosis. The disease is of sufficiently long standing that complement would be present in the patient's serum if a clinical coccidioidomycosis were present. Complement usually appears in about two weeks after the initial infections. Precipitins in this case may or may not be present. They are usually formed during the acute disease, and if present would indicate a recent active infection.

A *bronchoscopic examination* would also be indicated. It would aid in ruling out the possibility of a foreign body, mucous plug, intrabronchial disease, and extrabronchial compression.

Further sputum studies and gastric lavages, with animal inoculation and culture of the contents, are also necessary. Dr. C. E. Smith has suggested that in cases in which the tuberculin and coccidioidin tests are positive and the complement is not fixed, the only hope of diagnosis is by demonstration of the etiological agent, and there is no substitute for cultures for the coccidioides. Sabouraud's medium is usually used.

Other diagnoses that should be entertained but which are more unlikely are those of *bronchiectasis* and *virus pneumonia.* The factors predisposing to bronchiectasis, and the clinical history of the case, probably rule out the diagnosis of bronchiectasis. Lipiodol studies may be necessary to ultimately determine the state of the bronchial tree. A virus pneumonia is not out of the realm of possibility. While in the military service we did a series of lipiodol studies on patients having a diagnosis of virus and atypical pneumonias in which we found that intrabronchial changes did occur in some cases during and shortly after the acute infections. Subsequent recheck studies with iodized oil often showed a return to normal of the bronchial patterns. This raised the question of the etiology of bronchiectasis, but unfortunately, late follow-up studies on our cases were impossible.

Another diagnostic possibility is the presence of one of the tumors in the lymphoma group, with atelectasis from compression of the mediastinal nodes. A careful search should be made for nodes elsewhere in the body, with biopsy of one of these for confirmation. It is said that they are absent in the present case.

Pending a more definite diagnosis *the treatment should be conservative and supportive.* If coccidioidomycosis can be ruled out with a negative complement fixation test, the patient should be treated as a primary tuberculosis. Serial x-ray studies are warranted and, if progression is suspected either clinically or by x-ray, a course of *streptomycin* is justified. It has been observed that streptomycin is of value in the treatment of primary pulmonary tuberculosis, and it is also of definite value in the treatment of tuberculous adenitis. It is quite probable that a reduction in the size of the glands would relieve the compression on the bronchus if such is the cause of the atelectasis, but one would *not* use the drug, with its hazards, etc., unless definitely indicated.

No specific treatment is indicated for coccidioidomycosis. Bed rest and supportive treatment is advised, and the clinical course should be followed with blood sedimentation rates and x-rays. The chances are against the formation of a cavity, and also against a disseminated infection; there is good evidence that the two types of lesion almost never co-exist. Should cavity occur, conservative therapy is probably wisest, with lobectomy if the condition persists. When regression of the infiltration has occurred and the sedimentation rate has returned to normal, the child may be allowed to resume her normal activities. If the diagnosis of bronchiectasis is established, the most conservative treatment, considering the future, would be a lobectomy.

The prognosis in this case in view of the age and the probable diagnoses should be considered favorable.

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TOPICS OF CURRENT MEDICAL INTEREST

RX, DX, AND DRS.

By GUILLERMO OSLER, M. D.

Here is a piece of amazing news on epidemiology. Dr. S. W. Simmons of the U.S.P.H.S., an "economic entomologist," says that MALARIA can/will be wiped out in the U. S. within three years! . . . (Yellow fever was eliminated by a control of mosquitoes and patients, leaving some mosquitoes but no cases of the disease to serve as a source.) . . . Malaria is being controlled so quickly, by the use of DDT, that officials fear a let-up of effort in the 20 states in which it still exists. The incidence in Georgia, for example, has been reduced in the past two years from thousands of cases to a total of 10 cases in 1948.

Proof of the ARIZONA INTEREST IN ARTHRITIS was obvious on December 19th, when Dr. Edward Boland of Los Angeles spoke in Tucson at an open meeting. TWELVE HUNDRED PEOPLE jammed the U. of A. auditorium, many of them on crutches and in wheel-chairs. . . . Drs. Harry Thompson, C. E. Bensema, and Charles Stephens, Jr., also spoke on various aspects of arthritis. . . . The event accompanied the establishment of Tucson as the headquarters of the southwest chapter of the National Arthritis and Rheumatism Foundation. The area includes Arizona, New Mexico, Utah, Nevada, and West Texas. . . . A photographer for Life Magazine was on hand to report the meeting, which was the kick-off for a two million dollar fund campaign.

The hazy connection between PERNICIOUS ANEMIA, LIVER, AND THE VITAMIN B COMPLEX is slowly clearing—although the 20 years since it was first suggested are really a brief period. . . . Liver, and its extract, contain the substances needed to cure the deficiencies and results of pernicious and other hyperchromic anemias. Folic acid was recently thought to be the major factor, but it could not maintain certain cases nor reduce the degeneration in the spinal cord. . . . At present another member of the vitamine B complex (B12) promises to fill the gap. The exact composition has not yet been published, but it is said to contain cobalt, phosphorus, and nitrogen. It is a crystalline extract from liver, must be given by injection, but tiny doses are effective (1 microgram equals 1 U.S.P. unit of liver or stomach extract). . . . All subnormal cellular elements of the blood have been found to return to normal; the glossitis clears; the general feeling improves; and peripheral and central nerve changes rapidly improve.

Sometimes the membership of the STATE BOARD OF HEALTH seems to slip out of sight and mind. Since they represent both the medical profession and the public, their light should not be hidden for want of a place to show it. . . . The Board is composed of five members; each member serves five years; the Governor appoints a new member each year, so that the terms overlap. . . . The present personnel, and the duration of their terms, include Dr. Benson Bloom, Chairman (Feb. 1, 1950); Dr. Vivian Tappan, Vice-Chairman (Feb. 1, 1953); Emmett McLoughlin, Secretary (Feb. 1, 1949); Dr. Louis Baldwin (Feb. 1, 1952), and Mr. John A. Carollo (Feb. 1, 1951).

The ubiquitous ACNE VULGARIS, which deforms both the skin and the personality, now has an ingenious, bizarre therapy, described by a Detroit plastic surgeon (W. G. McEvitt). . . When the acute lesions have healed, the most notable bumps, pits, and sinuses are excised. The remainder of the surface is then SAND-PAPERED, under local anaesthesia, one or several times. The results vary, but may be dramatically good, and always show some improvement.

It is hard to know how cautious to be in printing early reports of medical progress. Here is one which is exciting, and is vouched for by one of the foremost men in the field. . . . Two Milwaukee physicians, Evans and Zeit, believe they have found the reason why certain substances (such as silica) cause PROGRESSIVE LUNG FIBROSIS, and others do not. . . . They have found that the reactive substances have non-symmetrical crystals, while the harmless ones are symmetrical. . . . Furthermore, the fibrosis forms, they believe, around the non-symmetrical crystals because they produce electrical energy when under pressure in the tissues, while the other crystals do not. . . . The logical next step is to look for a means to reduce or avoid the electrical charge.

The SANTA CRUZ GENERAL HOSPITAL ASSOCIATION has been re-organized on a permanent basis to assist the board of supervisors in arranging for construction of the $450,000 hospital which was authorized at a recent election. . . . One-third of the funds will probably come from the federal Hospital Construction grant. The site will be near Nogales. . . . Miss Jane Rider and Dr. James Ward of the Health Department have conferred with the group. . . . Among the 33 members of the Association are: Drs. Lee B. Milton, Charles Smith, Delmar Mock, Zenas B. Noon, J. S. Gonzales, M. I. Merritt, G. L. Harker, E. C. Houle and Peter J. Gekas.

The PROGRESS OF HOSPITAL CONSTRUCTION, under the Hill-Burton Act, is moving as rapidly as circumstances allow. . . . Dr. J. P. Ward, Director of Public Health, says that additions to the Maricopa County Hospital, the County Hospital at Florence, and the St. Mary's Hospital in Tucson have been approved. Gila and Santa Cruz Counties have voted bond issues which will probably be supplemented by federal funds. Casa Grande, Yavapai County, and Graham County are considering construction but have not yet issued bonds. . . . The federal allotment to Arizona for five years will amount to $2,260,875 and must be matched by twice that amount from the state. Early misunderstandings

of the allotment procedure were caused by failure to realize that committees had to be appointed, applications made, survey appropriations obtained, surveys made, federal standards of eligibility met, local politics and finances arranged, etc., etc.

The Atomic Energy Commission, through the Office of Naval Research, has approved 19 NEW PROJECTS for investigation, in the fields of biology and medicine in 14 American universities. This makes a total of 56 such projects under way at present.

The announcement that there were two new members of the AMERICAN COLLEGE OF SURGEONS in Arizona seemed like a good lead for a paragraph. The story, however, is too big—THERE ARE 78 MEMBERS OF THE SOCIETY IN ARIZONA! . . . There is a Fellow in Surgery in Douglas, Flagstaff, Ganado, Globe, Kingman, Nogales, Ray and Wickenburg; two in Cottonwood and Miami; three in Prescott, as well as a Fellow in Otolaryngology; 35 Fellows in Phoenix, including 18 in surgery, four in orthopedics, two in urology, one in thoracic surgery, one in gynecology and obstetrics, seven in otorhino-laryngology (five of which are also qualified in ophthalmology), and two in ophthalmology; and 26 in Tucson, including 13 in surgery, four in gynecology and OB, one in OB, two in orthopedics, one in urology, one in thoracic surgery, one in ophthalmology and ORL, and three who are listed as retired. . . . Quite a crop!

MOTOR VEHICLE ACCIDENTS produce many frightful injuries, comparable to those which resulted from battle and bombing. One of the major problems of both has been THE LOSS OF PERIPHERAL NERVE TISSUE. . . . Recent summaries of neurosurgery in World War II have shown remarkable hope. Defects in the major nerves of the extremities have been successfully repaired when the gap was 6 to 8 cm., and the theoretical maximum is almost twice that length. Methods which are used to obtain these results include mobilization of the nerves, positioning of the joints, and transposition of the nerves.

The war in China has produced some peculiar repercussions in Arizona. The dislocation of usual sources of OPIUM has spurred the development of a Mexican traffic. The border towns, and Phoenix and Tucson, become way-points and pay-off depots, and the population of undesirable characters and federal-men rises accordingly. . . . This situation probably does not affect the distribution of narcotic addicts, which is fairly high wherever there is chronic illness, and especially high in the offices of new doctors.

The use of GOLD THERAPY FOR RHEUMATOID ARTHRITIS must depend on the answer to two questions—Is it of value? and Is it hazardous? . . . There is little doubt that gold is the most valuable single method of treatment. The 'V' methods (vaccines, venins, and vitamines) are futile by comparison. It must be combined with physiotherapy and general care, but it is the leader. . . . Enough cases have been treated and observed long enough (Ragan of N. Y. Boland of Los Angeles, et al.) to define its indications, immediate effects, and late results. . . . The hazards are definite but not insuperable. Close observation, a knowledge of its effects, the use of insoluble preparations, and the use of BAL for detoxification can reduce the hazards to a low level. . . . Chrysotherapy was used quite early in Arizona, but the results were not summarized and published.

A Tucson newspaper, in trying to emphasize the decline of certain diseases, has tossed off the following gem of overstatement—"Tuberculosis has been pretty well conquered." . . . Still seventh on the list of killers, with more than 47,000 deaths last year in the U. S. The cause of 766 deaths in Arizona in 1947—by far the highest rate in the nation. The cause of disease in about 1% of the population. X-ray surveys have covered no more than 25% of our 148 million people, even once. Preventive measures, such as BCG and aseptic technic, have only slight usage so far. Beds for tuberculosis are in short supply in most areas, and notably in Arizona. Laws for control are rare and poorly enforced. Therapy is promising, and more successful, but has a relatively small application. . . . Progress is in the right direction, and the plan seems good, and we are hopeful—but certainly not over-confident

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Two points of interest about GASTRIC ASPIRATIONS FOR TUBERCLE BACILLI — False-negative results may be found if the specimens are delayed more than 10 hours before culture, especially in warm weather. Positive results in minimal disease were once thought to be rare, then found in 70%, later in 86%, and now in 97% of the cases (provided that recurrent tests are made, using all possible means of examination).

Clinical-Pathological Discussion*

From the Phoenix Clinical Club

DISCUSSANTS; Drs. W. W. Watkins, C. B. Warrenburg, and Kent Thayer.

An eighty-nine-year-old widow entered the hospital from a convalescent home because of oliguria.

The patient was unable to give a reliable history, and the only information that bore up under repeated questioning was that three weeks before hospital entry she had developed a cough productive of thick, yellow sputum and fever, nausea and abdominal soreness, with anorexia so severe that she took little food or water. Her physical disclosed that she had been given 120 cc. of Aldiazol, a liquid sulfadiazine preparation, over a two-day period. Four days before entry the urinary output dropped markedly, so that

* The case material used as a basis for these discussions is taken from the New England Journal of Medicine

only 180 cc. of urine had been passed in the forty-eight hours before admission. The patient had previously noted darkening and reddening of the urine, but the volume seemed to have remained fairly normal. She denied backache and dysuria. Although she had eaten little during this time, she had drunk considerable fluid.

The past history revealed that she had had shortness of breath on exertion but no anginal pain. For several years she had taken digitalis irregularly. She had long been constipated and had used laxatives regularly for more than a year. She had never had any children, the ovaries having been removed many years previously for an unknown reason.

Physical examination revealed a moderately obese woman with a dry tongue. Although the memory was poor, she was alert and co-operative. The chest was emphysamatous, with a moderate number of fine moist rales at both bases posteriorly. The area of cardiac dullness was obscured by emphysema. No murmurs were heard. There was slight tenderness in the right upper quadrant. The liver and spleen were not palpable. There was tenderness in both costovertebral angles, especially on the right. Pelvic examination revealed some tenderness on motion of the cervix upward. No masses were felt.

The temperature, pulse and respirations were normal. The blood pressure was 130 systolic, 65 diastolic.

Three cubic centimeters of dark-brown urine was obtained by catheter, and analysis showed a pH of 7.5, a plus test for albumin and no sugar; the sediment contained numerous fresh red cells, many epithelial cells and 1 or 2 white cells per high-power field. No bacteria or casts were noted. Examination of the blood disclosed a red-cell count of 4,100,000, with a hemoglobin of 10.5 gm., and a white-cell count of 24,000, with 91 per cent neutrophils. The nonprotein nitrogen was 133 mg. per 100 cc. The sodium was 137.3, the potassium 6.3, the carbon dioxide 31.0 and the chloride 95 millequiv. per liter. The blood was examined for sulfadiazine, but no trace was found.

X-ray examination of the chest showed cardiac enlargement, the cardiothoracic ratio being 13.23, but the configuration was not characteristic of any particular lesion. The wall of the aorta showed calcification. No fluid was present in the pleural sinus, and there were irregular areas of increased density at both bases. The pulmonary markings were everywhere a little prominent, but no gross areas of collapse or consolidation were seen. A plain film of the abdomen showed gas-filled loops of large bowel and of a portion of the small bowel, possibly ileum. Cystoscopy and retrograde pyelograms were negative. No urine, however, was obtained from either pelvis. Instilled fluid returned clear from the right pelvis and slightly blood tinged from the left.

During the next few days there was no improvement. The patient passed no urine, 30 cc. of bloody urine being obtained by catheter on the third hospital day. The fluid intake averaged 600 cc. a day. The costovertebral angle tenderness persisted, and nausea, muscular twitching and a positive Chvostek sign developed. The non-protein nitrogen rose to 154 mg. per 100 cc. On the fourth hospital day the patient expired.

DR. W. WARNER WATKINS:—

The past history indicates that some of her physical machinery was wearing out faster than others. The shortness of breath and the necessity for digitalis indicate some cardiac deficiency. The development of constipation in an old woman of sedentary habits, or possibly confined to bed, would not seem to be important. The immediate past history would indicate a respiratory infection, both on account of symptoms recited and because she was given a sulfa drug in fairly large doses. Aldiazol is a mixture of two sulfa preparations and she had 192 grains or 12 grams of this in two days.

The historical account is a little confusing and a correct chronology would seem to be essential in correctly evaluating the symptoms. We have the following facts to correlate:

1. Three weeks before the hospital entry this old lady developed cough, fever, nausea, anorexia, and abdominal soreness.
2. Four days before entry she developed oliguria.
3. Sometime during this three weeks, and presumably before the oliguria she was given the 12 grams of sulfa mixture in 48 hours.
4. She had "previously" (whenever that was), noted darkening and reddening of the urine.
5. The record states that in the beginning she took little food or water, and later in the same paragraph it is said that during "this time" (whenever that was), she drank "considerable fluid."

It would seem important to know:— (a) Just when in this three week period she took the 12 grams of sulfa; (b) whether she noticed the reddening of the urine before or after taking the sulfa; (c) whether the inability to take food or water occurred before taking the sulfa, or developed after that medication.

Whether this case was selected for that purpose or not, it is a good text on how NOT to use the sulfonamides, and I propose to discuss that matter in preference to making a diagnostic analysis.

There is no fool proof sulfonamide preparation. Some are more dangerous than others, but all require care and judgment in their use, because

any of them can result in severe tissue damage and sometimes in death. Any clinician who assumes the responsibility of administering any of the sulfa preparations should be familiar with the two types of damage which may lead to urinary suppression of varying degrees:

I. Mechanical damage due to the occlusion of the excretory pathways by crystals or concretions.

II. Chemical damage, due to toxic degeneration of the renal epithelium and tubules.

Before proceeding with sulfonamide therapy in anything approaching tolerance dosage, the clinician must observe certain precautions, if he has the proper regard for his patient's welfare. He must do the following things:

1. Determine previous sulfonamide medication, sensitivity or tolerance.
2. Determine the state of the renal function.
3. Determine the water balance.
4. Estimate and administer the proper dosage.
5. Maintain adequate alkalinization.
6. During and after the administration of the drug, keep track of the blood level of the sulfonamide and of the condition of the kidneys, by appropriate laboratory tests.

Let us briefly examine these requirements for safe use of the sulfonamides, as they apply to the case in question:

1. No information is given as to whether this patient had any previous sulfonamide therapy, or whether she had a sensitivity or idiosyncrasy to the drug, or whether any inquiry was made regarding this. Inquiry **should** have been made and the facts recorded, since severe damage or even death can result from hypersensitivity.

2. We have good reason to believe, from the record, that renal damage was present, not only that which is natural to such an advanced age, but we have the statement that the patient had previously noticed darkening and reddening of the urine. The laboratory tests recorded as having been done after the anuria developed, should all have been carried out **before** administration of the drug. Whenever there is a possibility of impaired kidney function,—and this is always not only possible but probable in an old patient,—the extent of this impairment should be estimated by the PSP test, urea clearance, ability to concentrate urine, and the NPN level of the blood serum. If the darkening and reddening of the urine did occur before the drug was given, this was a red light through which the doctor ran, and he should be given a ticket. If the symptoms of nausea, anorexia, fever and abdominal soreness were present before the drug was given, these also were siren warnings to the doctor to stop and investigate.

3. The determination of the water balance and its maintenance were also imperative. Because of the nausea, it is said the patient took little water or food. This would mean dehydration, a very dangerous condition in which to administer sulfonamide, because renal damage is more likely to develop in dehydrated patients. How to maintain the water balance is probably outside the province of a radiologist to discuss, but to paraphrase the famous message sent to the King of Spain by the enslaved Indians of Mexico—"we, too, are doctors," and it is easy to see from the record that this patient's water balance was not evaluated nor maintained, and sulfa administration invited further damage to kidneys already impaired by old age; it resulted in suppression of kidney function and eventual death, all of which should have thrown such a weight on this doctor's professional conscience as to sour his cocktails, make him miss a putt at golf or overbid his hand at bridge,—recreations to which he probably was giving more attention than to saving the life of this old lady. He might claim extenuating circumstances, in that she was already overdue as a passenger on Charon's ferry, and he just made certain she had a ticket.

4. In estimating the proper dosage of any sulfonamide preparation, the age of the patient must be taken into consideration. Older people do not excrete the drug as rapidly as younger ones, and this must be allowed for in setting up a tolerance dose. A dost of 12 grams would not seem to be excessive, if the precautions mentioned above are observed.

5. Adequate alkalinization is necessary, more so with some forms of the drug than with others. However, the administration of alkalies in cardiac or renal disease is almost as dangerous as omitting them when giving sulfa, and this patient certainly had cardiac disease and probably had renal disease. In such a situation, the physician needs to maintain a delicate balance and choose the lesser of two dangers.

6. During the administration of these drugs, if this extends over any length of time, or after its administration when it covers a short period of time, as in this case, determination of possible kidney damage at the very first evidence of reduced or impaired kidney function is imperative. If such determination was carried out on this patient before she entered the hospital on her last journey,—there is no mention of it in the record.

DIAGNOSIS

Toxic nephritis, urinary suppression and death,—resulting from professional delinquency in treating a patient with sulfonamide drugs. Contributing cause of death,—senile disintegration of the physical organism, including the kidneys.

DR. KENT THAYER:—

This patient undoubtedly died of uremia. The cause of the uremia seems to be the problem.

There are three general groups of causes of azotemia—post renal, renal and prerenal. I believe we can rule out post renal or obstructive because cystoscopy and retrograde pyelograms were normal.

Renal azotemia may be due to acute glomerulonephritis. It seems unlikely in the patient, although she did have a respiratory infection three weeks before the present episode. The urine showed one plus albumin and no casts. Red cells were present. One would expect more albumin, casts, and an elevated blood pressure.

Chronic nephritis also seems unlikely, for her blood pressure was normal and there should have been some casts present. The anemia should have been more severe, although the blood count probably was higher than it should have been because of the dehydration.

The uremia associated with Kimmelstiel's and Wilson's disease is due to chronic hypertension and diabetes. It seems our patient had neither.

Acute tubular nephritis caused by toxic substances such as mercury, transfusion reaction, crush syndrome and sulfadiazine can certainly cause severe azotemia. We have no history of any of these except sulfa. Our patient took 120 cc. of Aldiazol in two days. This contains five grams of sulfadiazine and five grams of sulfathiazole along with sodium citrate per 100 cc. of solution. Our patient ingested six grams of sulfadiazine and six grams of sulfathiazole in two days; this amount is certainly not an overdosage. We are not told how long before her oliguria this was taken.

We do know that her fluid intake was low from the onset of her acute infection which would tend to precipitate crystals of the sulfonamide in the kidneys. Patient noted darkening and reddening of the urine before the output decreased—evidently bloody urine. The fact that sulfadiazine was not found in the blood at the time of admission into the hospital does not rule this drug out.

Sulfonamides may be precipitated in the tubules, and not only cause obstruction in the tubules, but also degeneration of the tubular epithelium. The crystals may be found in the urine, but no mention was made of this in the protocol. Alkalizing the urine is said to prevent, to a certain extent, this precipitation. However, while in San Francisco last spring, one speaker tried to show how soda bicarbonate would increase this precipitation process and warned against its use. Fluid intake probably has a more determining factor.

In the service, the emergency first aid packets contained four grams of sulfadiazine for the injured man to take as soon as possible after the accident. In some of the warmer climates this was discontinued because of the decreased urine output because of sweating, and the increased number of patients with renal damage due to these drugs.

Prerenal azotemia is usually caused by peripheral circulatory failure, the causes of which are several:

1. Liver and biliary diseases such as cirrhosis of the liver, or acute yellow atrophy, or ascites. We do not have evidence of any of these in this patient.
2. Vomiting or diarrhea of long standing will dehydrate patients severely and the urine output is decreased.
3. Shock and circulation collapse, or severe hemorrhage may cause azotemia.
4. Coronary thrombosis. The urine output is decreased if the occlusion causes rather severe heart damage.
5. Terminal heart failure is nearly always associated with an elevated blood NPN because of an insufficient circulation. Acute intestinal obstruction is associated with an elevated NPN because of dehydration and the circulatory collapse.

I have been unable to fit any of this to our patient. We know a coronary occlusion may be "silent," but to cause this severe uremia, there must be more evidence of heart failure.

It is my belief that our 89 year old lady developed a respiratory infection and bronchopneumonia as shown by the roentgenogram. With the onset of this she stopped drinking water. Sulfonamide drug was given which was stopped in the kidneys, producing a tubular obstruction and degeneration resulting in anuria. The renal tenderness and blood in the urine fit very well. The patient went on to uremia with nausea, muscular twitching, and death.

DR. CLARENCE B. WARRENBURG:—

This 89 year old lady is no doubt well packed with pathological specimens. Indeed, there are many people who feel that it is pathological to reach the age of 89 in the first place.

We are confronted with the problem of determining the particular pathological condition, or conditions, that caused this patient to die. She was admitted to the hospital because of an oliguria which finally amounted to an anuria.

The history of having taken 120 c.c. of aldiazol over a 48 hour period prior to her urinary "shutdown" is, of course, vital information. In prescribing medication for infants and the aged it must be remembered that their tolerance for drugs is not that of people in the second and third decades of life.

Aldiazol is a preparation which contains 0.25 gms. sulfadiazine and 0.25 gm. sulfathiazole in each five cubic centimeters, with sodium lactate and citrate added as alkalinizers. Our patient took 12.5 gms. of these sulfonamides during a 48-hour period, which is considered to be the average adult dose; but as mentioned a moment ago this patient is almost 90 years old.

I found this interesting bit of information about the sulfa drugs: Lehr, Slobody and Greenberg[1] have advised the use of a sulfadiazine, sulfathiazole mixture in an effort to lower the incidence of mechanical renal difficulties. They base their concept as follows: the precipitation of sulfonamides in the urinary tract is largely dependent upon the solubility, and the concentration of these compounds in the tubular urine. Solubility studies with sulfonamides revealed that sulfathiazole, sulfadiazine, and sulfamerazine, for example, could be dissolved **simultaneously** in the same sample of water or urine to the point of full saturation of each compound when present alone. The concentration of these 3 drugs in a saturated solution represented, therefore, the sum total of their individual solubilities. It was reasoned that the danger of intrarenal precipitation from the drugs comprising such a mixture should only be as great as if each compound had been administered alone and in the partial dosage contained in the mixture. The validity of this conception was substantiated in animal experimental studies as well as in clinical trials. Therefore, our patient today actually received a little over 6 gms. of sulfonamides; as far as precipitation in the urine is concerned. Being mindful of this relatively small dose it is still my feeling that this was the trigger mechanism that set off the chain of events that led to this patient's "untimely" death. The history mentions that the patient had previously had "darkening and reddening" of the urine. It seems logical to rationalize that a sub-clinical glomerular nephritis or nephrosclerosis was present, and following the administration of sulfonamides with precipitation of the crystals in the urinary tubules a fatal renal insufficiency developed.

The patient died a uremic death. The costovertebral tenderness, the oliguria and hematuria and an NPN as high as 154 mgm. per 100 c.c. of blood undoubtedly speaks for grave kidney damage. The acidosis, dehydration, and evidence of disordered calcium metabolism, as manifested by a positive Chvostek sign, add further evidence of advanced renal insufficiency.

Hypertension and edema are usually found in chronic glomerular nephritis and nephrosclerosis, but not invariably. Such findings are not present in this case.

From the records of the Massachusetts General Hospital:

DIFFERENTIAL DIAGNOSIS

DR. CHARLES L. SHORT: The first question that we must answer is, What was the nature of the original infection that apparently began three weeks before admission? This was presumably in the respiratory tract and suggests bacterial pneumonia. Perhaps we shall be helped by the x-ray films of the chest. May we see them now?

DR. STANLEY WYMAN: I do not believe that the films will be helpful. They show a large heart and a few, small indefinite linear areas of density in both bases, which, I suppose, lie in the lower lobes. I do not see frank consolidation.

DR. SHORT: Does that picture suggest infection rather than congestion?

DR. WYMAN: It is the picture one sees in atelectasis in any patient lying in bed with some impairment of diaphragmatic motion.

DR. SHORT: Does the film of the abdomen show any definite point of obstruction or rather suggest ileus?

DR. WYMAN: The film of the abdomen is more in keeping with ileus than with true intestinal obstruction. The retrograde pyelogram shows good calyxes; pelves and ureters on both sides.

DR. SHORT: There is no definite evidence of pneumonia at the time of admission. We also have nothing to suggest any marked degree of pulmonary edema. The patient had no fever when she entered the hospital, which suggests that the infection, whatever it was. had cleared. The high white-cell count can probably be explained by a renal injury.

The next question and the chief one to answer is, What was the cause of the anuria and uremia that were evidently responsible for this woman's death? I am unable to decide from the evidence at hand that any of the usual forms of nephritis were present, although an acute glomerulonephritis secondary to infection could produce a clinical picture somewhat similar to this. The lack of fever is against a suppurative lesion of the kidney, and such rare conditions as dissecting aneurysm involving the renal arteries and renal infarction, I believe, we need mention only in passing.

I am forced to conclude that the renal shutdown was directly connected with the sulfonamide preparation taken by the patient. This preparation, I have been informed, contains 1.5 gm. of sulfadiazine and an equal amount of sulfathiazole per 30 cc., with additional sodium citrate and sodium lactate in an aqueous solution, so that we do not need to worry about a toxic solvent's being involved. In the dosage given this patient received 6 gm. of each drug, which does not seem excessive over two days, and we know that the solubility of the drugs was probably enhanced by the mixture of equal parts of each drug and by the additional alkali. For this reason, as well as the lack of severe pain, the absence of crystals in the urine and the negative findings on washing of the kidney pelves, I have decided against obstruction of the kidney pelves or the ureters by sulfonamide concretions as the cause of the anuria. These considerations do not rule out obstruction in the tubules themselves by sulfonamide crystals, but I prefer to believe that the anuria was due chiefly to a direct toxic action on the renal parenchyma. Such a process has been clearly described pathologically without evidence of obstruction by crystals, although it is less frequent as a cause of sulfonamide renal impairment than obstruction.

I might mention still another possibility: that the renal damage was due to an acute hemolytic anemia resulting from sulfonamide administration. These cases occur nearly always with sulfanilamide, and we should expect jaundice and a severer degree of anemia to be present. As a matter of fact, if such an anemia had occurred the pathological findings would have been closely similar to those that I expect to be found in this patient.

Before concluding, I should like to attempt to answer a few more questions. It would be interesting to know whether or not previous administration of sulfonamides had taken place, so that the severity and the abruptness of the reaction could be explained on the basis of hypersensitivity. We are not helped by the history, and there were no other manifestations of sensitivity, such as fever and dermititis. It is still possible that focal lesions in the heart and other organs will be found or even vascular changes resembling periarteritis nodosa, as described by Rich.[1, 2] But the short duration of administration of the drugs makes this unlikely unless the patient had previously been sensitized. Whether or not she had pre-existing renal disease that had made her especially susceptible to sulfonamide intoxication, I do not believe that we can find out, although the negative pyelogram helps rule out certain anomalies and diseases of the kidney. The site of damage in the group to which I think this patient belonged is usually the distal portion of the tubules, with involvement of the interstitial tissue also—so-called "lower-nephron nephrosis." A similar clinical picture can be produced by cortical necrosis. I have seen a reference to this condition that was presumably caused by sulfonamide intoxication,[3] but I do not believe that we need consider it in the case under discussion.

In summary, I believe that the primary cause of death in this patient was uremia resulting from tubular necrosis caused by sulfonamides, with cardiac enlargement and arteriosclerosis as additional diagnoses. I do not believe that I can say anything about the changes in the lungs, unless edema developed before death. In a woman of this age other abnormalities or landmarks may well have been present. but I shall not try to predict what they were.

DR. LOUIS K. DAHL: In general the house staff believed that the diagnosis rested between three main possibilities: acute glomerulonephritis, with sudden shutdown; interstitial nephritis due to sulfonamides; and—one person thought—bilateral cortical necrosis. The most prevalent opinion was interstitial nephritis caused by sulfonamides.

Clinical Diagnosis

Interstitial nephritis caused by sulfonamides.

Dr. Short's Diagnoses

Lower-nephron nephrosis, due to sulfonamide toxicity.

Cardiac enlargement.

Arteriosclerosis.

Anatomical Diagnoses

Periarteritis nodosa.

Diffuse cortical necrosis, secondary.

Arteriosclerosis.

Hypertrophy of heart.

Pulmonary emphysema.

Cholelithiasis.

Pathological Discussion

DR. MALLORY: Autopsy showed interesting kidneys. They weighed 450 gm., and for a woman of eighty-nine that is significant hypertrophy. The surfaces were mottled and purplish red, and the capsules stripped fairly readily. When the organs were cut it was found that the entire cortex of both kidneys was infarcted, presenting the gross picture of diffuse cortical necrosis. This is seen most frequently—in about two out of every three cases, I should say—in young women during the terminal stages of pregnancy or in the first few days following delivery. At the age of eighty-nine that possibility need not be considered. Cortical necrosis has been seen in a scat-

We do not have the references 1, 2 and 3 in the library.

tered variety of other conditions and is occasionally seen in male patients.

When we examined sections of the kidneys microscopically another possibility was brought up. In all sections there were many small arteries showing the typical changes of periarteritis nodosa. A few arteries outside the renal substance also showed the same lesion—few indeed, but some. There was a characteristic lesion in one adrenal gland, a few changes in the perirenal fat tissue, and a single lesion in the heart. Diffuse involvement of the small renal arteries with periarteritis nodosa might produce the same total infarction of the cortex that one sees in so-called "primary cortical necrosis." I think that the way in which this case should be classified remains debatable. The facts that the arterial lesions were so characteristic of what one sees in periarteritis nodosa and that at least a few lesions were found in organs other than the kidneys lead me to make periarteritis nodosa my primary diagnosis.

The other changes found were only those that one might expect to find in a very old person,—extremely severe arteriosclerosis of the aorta, with multiple ulcerated atheromas and secondary thrombi,—but the main renal arteries were not markedly involved. The heart was hypertrophied, and the lungs were emphysematous; the gall bladder was filled with small stones, and the bile ducts were patent.

DR. SHORT: What did the tubules show?

DR. MALLORY: The glomeruli and all tubules in the cortex were completely necrotic, a picture exactly like that of infaction, except that it was symmetrical and diffuse throughout the entire cortex of both organs.

DR. FLETCHER H. COLBY: Were you able to determine which arteries were responsible?

DR. MALLORY: In cases of idiopathic cortical necrosis, vascular changes are variable and inconsistant. There are usually necrosis and thrombosis within the actual areas of infarction, but vascular lesions are not found outside. Not all cases show vascular changes, so that it is difficult to attribute the cortical necrosis to a primary mechanical obstruction of the arteries. The pathogenesis of primary cortical necrosis remains a complete mystery.

DR. COLBY: Presumably toxic?

DR. MALLORY: A similar lesion has been produced in experimental animals with staphylococcus toxin, but in human beings there is rarely evidence of an infectious etiology. In this case, the finger of suspicion points strongly to sulfonamide sensitivity. It is interesting, however, that no trace of persisting sulfonamide, either free or acetylated, could be found in the blood plasma.

MEDICAL LEGISLATION AS VIEWED BY A CONGRESSMAN

By HON. FOREST A. HARNESS,

LADIES AND GENTLEMEN: It is a genuine pleasure to meet again with a representative group of your honored profession.

On numerous occasions in the past fifteen months, it has been my privilege to discuss with representative medical groups the work of my Committee on Publicity and Propaganda in the Executive Departments. I have reported in detail to your profession the tremendous forces within government which seek day by day to condition mass thinking, to foster national policies conceived within federal agencies, and to perpetuate and enlarge federal bureaucracy. Your profession has been particularly interested in the first major disclosure which this committee made.

I know each of you is painfully aware of the mounting threat of compulsory health insurance or political medicine, as I choose to call it. You are familiar with the pattern of federal propaganda in this field, for the revelations of the committee have been quite widely publicized by the National Physicians Committee and other representative groups and organizations within your profession. It is unnecessary to repeat in detail here the disturbing details of the story.

It is sufficient to say that six agencies within the Federal Security Agency have wrongfully expended public money and misdirected the talents and energies of a large group of federal executives and employees for the specific purpose of convincing the American people that national health should be the sole responsibility of federal government.

Furthermore, the real nucleus, the driving spirit, in the campaign to socialize American medicine lie within this sprawling branch of federal government. I sincerely believe that my committee has made a real contribution to the American people by revealing at least the broad outlines of the federal propaganda machine and by showing at least in a general way the nature and immensity of its operation. Unfortunately, however, the 18 months in which we have operated have been sufficient only to write the introductory chapter of the detailed story.

Of course, you would like to know what to expect of the future. I have always hesitated to make predictions, and I am sure that if I had ever fancied myself as a prognosticator, the horrible fate of the pollsters and the political soothsayers on November 2 would deter me now. The fate of this committee, however, hardly

Speech of Hon. Forest A. Harness, Chairman, House Committee on Publicity and Propaganda, before the Conference of State Medical Association Secretaries and Editors, St. Louis, Missouri, Sunday, November 28, 1948.

seems to be in question. I have no doubt that when the 81st Congress is organized under Democratic control the serious organized effort to expose and to curb federal propaganda activities will cease.

That brings us to a question of the most vital interest to every one of us here. Can we, as individuals working through private organizations, effectively carry on the work which we had hoped would be consummated in the 81st Congress with the sincere cooperation of a new administration? For my own part, I believe we can, if we are willing to devote the necessary intelligence and energy to the task.

Although I have been denied the opportunity to serve in that body, it shall be one of my principle objectives to see that this work is carried on informally in the 81st Congress by the Republican minority. In every way possible, also, I shall devote my own efforts to the cause.

I know that you and a preponderant majority of the medical profession as a whole share my views and desires. For that reason, I shall take the liberty to speak very frankly to you and to say a number of things which I warn you will not make pleasant listening.

First of all, let us face the disturbing fact that the entire world has been, and is, politically sick. Let us recognize that this insidious, all pervading illness has made serious and possibly permanent inroads in America. There are a hundred factors, big and little, which influenced the decision which the American electorate returned on November 2, and only the historians will be able to appraise these factors in the order of their importance.

One fact, however, does stand out in painful clarity. A majority of the American people obviously have come to embrace the philosophy and policies of the powerful, highly centralized, paternalistic type of government which we have experienced in the past sixteen years. That is as hard for me to believe and accept as it is for you.

In fact, so long as we stay in the rarified atmosphere of academics, a thousand polls will prove that it just isn't so. In pure theory, a preponderant majority of American people believe in our free institutions, as opposed to any form of state socialism. But the minute we go from the theoretical to the practical level, we encounter a thousand selfish special interests which sway individual and group thinking. And here is the key to our failure.

As simple examples: American consumers vigorously reject the theory of rigid controls and rationing, for the reason, as President Truman himself pointed out in 1946, that these are the methods of the police state. Yet, in an emergency of inflation such as this, great numbers of them are persuaded to accept controls as the only protection against burdensome prices.

Producers, who even more vigorously resist the theory of domination by the state, are willing in too many cases to submit to what they hope will be moderate and temporary restrictions in the belief that government can best insure them continuing high prices and profits.

Never mind the fact that these people are deluded. We know that. What matters is that they do not know it, or refuse to admit it.

Incidentally, if there is any single, oversimplified reason why the poll takers and political forecasters were so grossly wrong in their predictions of this election, it is that they failed to find or to apply practical measurements at street corner level. As a practicing politician, I confess regretfully that I failed about as dismally as the rest in my own appraisals.

With this confession of my own failure, let me point out some of the more serious failures and short-comings within your own profession. At the academic level, I know medical people have made tremendous efforts, both to resist the encroachments upon free institutions, and to meet the growing threats to your own profession. Through such agencies as your National Physicians Committee you have made important investments of time, money and effort in the fight for the free institutions in which you believe.

Those investments have certainly been worth while, if only for the reason that they have strengthened the resistance in past congresses—both Republican and Democrat—to the drive to socialize American medicine.

But what effective effort has the profession ever made at the practical level? How much time, effort and substance have you ever devoted to everyday, down-to-earth, shirt-sleeve politics? Can you recall a single instance when the profession, or significant numbers within it, vigorously endorsed and supported a political party, or individual candidates?

You have spoken for our ideals, to be sure. But I regret to tell you that you simply have

not peeled off your coats and fought with, and for the candidates who carry our battle flags. You indulge the mistaken notion that the profession must not sully itself by stooping to political activity. The letterhead of your National Physicians Committee emphasizes your aloofness by describing the organization as "non-political."

Change that to "non-partisan" if you like. But try to adjust your thinking to the inescapable fact that trends are not started or stopped in council chambers, but on street corners. Efforts expended at academic levels, and possibly directed to Congress at a given time, may speed or retard a trend. But if the efforts are to be permanently effective, they must reach the masses who write the decisions each two years in national elections.

I think it is a great pity that people like you, who are best fitted by ability and training to provide leadership, have remained aloof. I think it is particularly regrettable that our medical people, who enjoy positions of confidence and trust hardly equalled by any other group in the American community, have so far neglected to prescribe for the body politic as part of their daily ministrations.

Your course in the future is naturally a question which only you, yourselves can answer. As a layman, who has spent many years in the business of everyday politics, however, let me urge that you are going to have to fight more practically and effectively for the things in which you believe, if you expect to preserve them. In this connection, I would like to make a number of specific recommendations to you.

The first of these is that you find the means to improve your public relations. The history of medical advancement in the United States is little short of miraculous, but have the American people been made to understand that fully? Does anyone take the trouble day by day to hammer home to the people how vastly superior our medical services and facilities are to anything anywhere else in the world?

Has anyone devoted even a fraction of the effort put forth by the advocates of state medicine to an honest, realistic, convincing portrayal of the failures and short-comings of political medicine as it has been tried elsewhere in the world? Is anyone trying to give the American people a true picture of the current tragedy in Great Britain as state socialism engulfs your profession, and sets up a system of political medicine?

The medical profession suffers a chronically bad press, due to the constant flood of reports, many coming from government sources and paid for by your own money, of the weaknesses and short-comings of the profession as it functions under private guidance. Day by day these forces remind the American people that medical and hospital care is beyond the reach of lower income groups, that vast rural areas of the country are provided woefully inadequate services and facilities.

Continuously, insidious implications of professional selfishness and profiteering are pumped out to the people in a way which certainly will have devastating long-range results if not effectively counteracted.

As members of the profession, you undoubtedly are aware of this unfortunate condition and probably feel it and resent it more keenly than most. I hardly need to point out hundreds of specific examples which recur almost daily in press and radio.

If you feel resentment, let me urge you not to misdirect it at your newspapers and radio stations. They don't make the news. They merely communicate it. When an important agency of government, such as the Federal Security Agency, acts, its action is newsworthy. When prominent public officials engineer a nation-wide meeting, such as the National Health Assembly, held a few months ago in Washington, they create a public sounding board which can be used with tremendous effect.

Such events, such activities, are newsworthy and never mind the fact that the news is uniformly bad from our point of view. The unfortunate fact is that bad news is usually more spectacular than good. A murder case will hit the front page on the same day that a dozen life savings by surgeons and physicians will appear as modest, routine items, buried in the ads, if indeed they make the paper at all.

The disaster at Pearl Harbor was hardly pleasant reading or listening, but probably nothing that has ever happened in the life of any of us so completely filled newspapers and radio. The unusual and the spectacular make the headlines because they are just that. The commonplace seldom comes to attention, and you in the medical profession can be proud of the fact that competent medical care in America is com-

monplace. But there is much more than that in this problem.

A meeting of the American Medical Association will be covered just as fully as this National Health Assembly or some similar activity designed by the medical propagandists. The latter will probably rate more publicity, however, because it will emphasize the problems, the failures, and the goals still to be attained, not the amazing services and facilities enjoyed so long that the average person takes them for granted.

And here is another distinction which I cannot possibly emphasize too strongly. A private medical group or organization will conduct a meeting or announce an advancement which momentarily proves as newsworthy on the constructive side as anything the propagandists ever do destructively on a single occasion. But the doctors meet, conduct their business, make their announcements, and go home until the next annual event.

The propagandists, however do not disband, content with the banner headlines they achieve at any given moment. Instead, those headlines become grist for the propaganda mill which grinds 365 days of the year. The private organization goes quietly back to its own business. The propaganda organization goes back likewise to its own business, but note the vast difference. Your business is medicine. Theirs is to sell ideas.

If we can criticize the newspapers and radio for anything at all, it is for their sentimental weakness in human interest stories. In this field they unwittingly work frequent injury to your profession in a manner which I am afraid the average physician or surgeon doesn't realize.

Let me show you a little case in point in the District of Columbia. Several months ago, a young woman was shot in a Washington nightclub of very doubtful reputation by a drunken police officer. The bullet struck the spine and the young woman has since been paralyzed from the waist down. Note that here are all the elements of spectacular news which put the story in headlines in every Washington paper. It subsequently develops that the victim may possibly be restored by surgery of a high order and by prolonged hospitalization. Here, obviously, is a natural for newspaper development. A spectacular story in the first place, an unfortunate, innocent victim without funds, and a human interest promotion angle which a Washington newspaper readily sensed and grabbed.

As a result, a feature writer for that paper is beating the drums for subscriptions and will almost certainly raise the funds to defray the costs involved in this young lady's recovery. I presume all of you have seen this Washington case duplicated in its essentials one hundred times, but I wonder if any of you recall a single instance in which the medical profession itself grabbed the ball and earned the credit which eventually accrues in such cases.

The code of your profession decries press agentry and sensationalism. I have nothing but commendation for your motives in this respect. I know that physicians and surgeons throughout the United States daily perform services of a purely humanitarian nature equalling the case in point. And seldom are the stories ever told, because most doctors actually avoid publicity.

But why should not our doctors dominate public interest stories like this? Why should the medical people shrink in the background particularly when their reticence opens the way for unfortunate implications against the profession? And just look how such publicity implicity hurts the private practitioners.

A spectacular case develops. It makes headlines, and fixes an individual victim's name in the public mind. Then public subscriptions are started. WHY? Here comes the dangerous inference: The victim is just another one of the millions of poor, deserving people who cannot afford adequate medical and hospital care.

Being no public relations expert myself, I am not prepared to suggest an effective remedy. If I were responsible for the future status of the profession, however, I believe I would take the problem to the best public relations specialists available and would move heaven and earth to apply the remedy these specialists might suggest.

In discussing the following specific recommendations to the profession, let me admit at the outset that it is presumptuous of a layman to intrude in problems which only the profession can solve. I do presume to discuss these problems with you, however, because each of them individually and all of them together do have serious political implications. They are, in fact, the principal ammunition of the forces which are pressing to nationalize medicine in the United States.

The first and most basic question is perhaps that of making adequate medical and hospital care available to all income groups. Patently,

there are vast numbers of people at the lower end of the income scale who are able by their own means to pay for nothing more than the barest emergency services. While that is certainly no fault of the profession, it must be accepted as a primary responsibility of private medicine, if we are not indeed to dump the problem into the lap of government. For, believe me, these problems which we cannot, or will not, solve for ourselves through private effort at state and community levels will go by default to Washington for some solution, good or bad.

The bare statistics would indicate that the medical profession is making progress in supplying the trained personnel to meet national medical needs. The figures tell us that we have roughly 17% more doctors in all categories than we had in 1940, while the population increase in that period has been approximately 12%.

I frankly do not know what significance there may be in these comparative figures, however, for I suspect that the total number of doctors includes a far more than normal number of practitioners who are far over effective age. I am sure, too, that included in that number are a vastly increased group in institutional practice.

For proof it is not necessary to break down the statistics because we know the military services and the Veterans Administration alone are employing many times the number of doctors they had in service in 1940. The number of doctors in effective private practice per capita, therefore, is probably somewhat under the figure for 1940.

Certainly the medical profession cannot be blamed for the violent dislocations which occurred in the war. We suffered a serious lag in all types of professions and technical training in that period, and we shall inevitably pay for it in the medical field as in many others.

But the alarming fact remains that we have done little, if anything, to date to compensate for that serious lag. I understand, in fact, that there are today only 1500 more medical students in training than there were in 1940. That increase is barely adequate to meet population growth. It will not make up for our war-time lag. And it certainly will not carry us through a national emergency, such as another war.

Here again, it certainly isn't the sole responsibility of this profession to expand the medical training facilities. But who else but government will supply the solution if the profession does not do so? In the meantime, I regret to tell you, the medical profession generally today is being criticized on the assumption that it is responsible for maintaining unrealistically rigid training standards and unnecessarily hampering the expansion of training facilities.

I frankly have no answer for the problem of expanding these facilities to meet immediate and future needs, but I do devoutly hope that the answer will come from the profession and the private schools, rather than from a political bureau of medicine in Washington.

The question arises in the same pressing manner for technically trained personnel, such as nurses, and for hospital facilities. And it seems alarming to me that we have only approximately 1000 more nurses and 200,000 more hospital beds in all categories today than we had eight years ago. Shall we find private solutions, or shall we let the problems go by default to medical bureaucracy?

Now about the distribution of services and facilities. And here the disparity in many cases is actually alarming. The factors which motivate medical men are, of course, perfectly natural and understandable. It is easy to see, therefore, why the average young fellow entering private practice gravitates to the metropolitan areas and the wealthier communities, while large rural areas and small towns drop lower and lower in the comparative scale of services.

I know the profession itself is as deeply concerned about this increasingly serious situation as I could possibly be as a layman. I know, too, that it has sought by one means and another to attract young doctors into these areas which are inadequately served.

Frankly, however, the profession's efforts to date have been totally inadequate. And here again we may as well recognize the unhappy fact that unless we find solutions through private effort the pressure for a Washington solution will mount. We have simply got to find the means at state and local levels to provide adequate hospital facilities and trained technical personnel which will give the rural and small town young doctor the tools with which to work effectively and the incentive to build his career in these areas.

If this were merely a layman's discussion of these technical and professional problems, I would apologize for wasting your time. For you people know all of these problems much more in-

timately than I do. And you are much more capable of solving them professionally than I. I mention them here only because I want to emphasize as strongly as I possibly know how the fact that there are the most serious political implications in everyone of them. I want to emphasize again and again that no one of these problems in itself, nor the overall problem of maintaining independent medicine in the United States is going to be solved at the academic level. And that brings us back to our point of departure. The medical profession faces an unhappy—indeed, an alarming, set of conditions. The world is sick, and the malady has reached the American body politic. The preventives we have so far applied simply have not proved strong enough to halt the inroads; the mild remedies have not effected recovery.

I would suggest that it is high time to try something more direct and drastic. I believe the medical profession faces the necessity of rolling up its sleeves and going to work right on the street corners of America.

If you hope to survive as practitioners of a free and untrammeled science, you must, in my opinion, become practicing politicians in the same sense that the organization members and the candidates of a political party learn to practice the art of politics around the clock, 365 days a year. If you continue as academicians, as in the past, I frankly fear that you will soon practice as a medical bureau in Washington directs you.

I am speaking to you in complete frankness, and with only the most sincere desire personally to forestall the tragic eventuality we are all fighting. I have said some unpleasant things here about the medical profession, but my remarks apply not alone to doctors. I have pointed them specifically only because this is a specialized group. But they might apply equally to all professions, and all those people in business and industry who have so far refused to come directly to grips with the forces which threaten our free institutions.

I purposely point these remarks to medical people, for I know of no point at which the basic issue is more sharply joined than in the field of medicine. Public health—and some form of socialized medicine—is invariably a focal point, a spear-head of any drive for state socialism. It is readily dressed in the robes of noble purpose. It has mass appeal. Therefore, it is the handiest possible weapon of would-be dictators, and of bureaucrats who seek to perpetuate themselves in power. Give a government agency the authority to supervise the daily health problems of every citizen, and to handle the money for national medical and hospital care; and you put government further into the lives of the American people by a single step than it has ever encroached in the entire course of our history as a nation.

In no other manner, and by no other devise, short of actual violence, could America's free institutions be more quickly destroyed.

Well, just how do you feel about the immediate prospects? There certainly is nothing in the present picture to warrant optimism. I imagine that medical people today feel about as glum as all the rest of us who have fought with little apparent effect to stem the tide of state socialism in recent years.

Still, this is a fight we cannot abandon—one we must not lose.

And if we need new inspiration to carry on, we can find it abundantly here today in the history of your profession in America.

I said at the outset that your history of progress in this nation is little short of miraculous. That continuing miracle, of course, has been made possible only by the greater miracle of individual liberty and opportunity under constitutional representative government. I know of no other group so fully conscious, and so deeply appreciative, as your own profession. For that reason, I confidently expect you to be in the vanguard as we continue the fight to restore and preserve our precious heritage.

TWENTY-FIRST ANNIVERSARY YEAR OF HAROFE HAIVRI

The Hebrew Medical Journal Volume II, 1948

With the appearance of Volume II, 1948, THE HEBREW MEDICAL JOURNAL, edited by Moses Einhorn, M. D., concludes its 21st successful year of publication.

In publishing the Journal, the editors aim to meet the need for a medical journal written in Hebrew, with English summaries, thus aiding greatly in the advancement and development of Hebrew medical literature.

This issue contains an article on Hypertensive Vascular Disease by Benjamin Jablons, M. D. There is also a discussion on clinical observations

and treatment of 190 cases of Malaria in Palestine, by Dr. P. Ephrati of Tiberias.

In addition, under the heading of "Personalia," biographical sketches of Professor Heinrich Finkelstein, great pediatrician; Professor Max Neuburger, renowned medical historian; and Dr. Solomon Solis-Cohen of Philadelphia, are presented.

For further information, communicate with the Editorial Office of THE HEBREW MEDICAL JOURNAL, 983 Park Avenue, New York 28, N. Y.

Cancer Current Literature

Shellito, John G.; and Dockerty, Malcolm B. CARTILAGINOUS TUMORS OF THE HAND. Surg. Gynec. and Obst. 86:465-472 Apr.) 1948.

"Cartilaginous tumors of the hand, on the basis of pathology, may be divided into two types: enchondroma and ecchondroma. The synonym for enchondroma is chondroma. Ecchondroma is called 'perichondroma,' 'osteochondroma,' and 'exostosis.' (The word 'osteochondroma' is restricted to the ecchondroma.) The fact that the entire group of cartilaginous tumors is commonly called osteochondroma has led, both in the past and in the present, to a great deal of confusion. It is better to use the terms enchondroma and ecchondroma.

Distinction between the two cannot be made with slide and microscope alone.

An enchondroma is usually a small cartilaginous tumor located centrally in the bone shaft, growing from many centers and expanding in all directions. Rate of incidence of pathologic fracture in our series was 26.6 per cent. The enchondroma occurs much more often in the hand than does the ecchondroma (3:1).

These tumors, in comparison to normal hyaline cartilage, show increased vascularity, calcium deposition, and myxomatous degeneration.

Two tumors of the tendon sheaths were found, both of which were enchondromas. In one of these cases multiple enchondromas of the sheath had developed and this is good evidence that an enchondroma can occur in the tendon sheath away from the site of tendon insertion.

The ecchondromas occur peripherally in the bone near the end of the shaft and have a bony base and cartilaginous cap.

Either type of tumor may be malignant, in which case it is usually a sarcoma. In this series 2 per cent were malignant. Both types are prone to recur although recurrence does not mean malignancy unless a mitotic figure can be found.

The treatment in both types is conservative operation." (Author's Summary)

24 references, 4 figures.

Langley, W. D.

MULTIPLE MYELOMA. Bull. Guthrie Clin. 17:179-183 (Apr.) 1948.

"Multiple myeloma is a rare disease characterized chiefly by back pain, weakness, anemia, and changes in the bony skeleton in 80 per cent of the cases. It is a malignant disease whose overall picture may vary tremendously, which fact has caused some dispute regarding its true nature. It appears that the abnormal cells arise from the reticulo-endothelial cells. It is a fatal disease with death occurring in about 18 to 24 months of the onset of the symptoms, although there are cases on record of patients surviving for longer periods. The use of diamidine compounds seems to offer these patients a chance of longer survival while at the same time controlling the most annoying and commonest feature, pain.

The disease should be suspected in patients in the middle and later years of life who present an unexplained anemia or generalized osteoporosis in whom no apparent cause can be found, or in those patients who are thought to have an undetermined primary malignancy with bone metastasis, in those problems with compression fractures of the spine from mild trauma and finally where backache is an annoying problem and no cause is found. For after all, a high percentage of patients with multiple myeloma present backache, anemia, and progressive weakness as the cardinal symptoms.

Various laboratory procedures have been employed to further evaluate the problem of multiple myeloma but we were unable to draw any significant conclusions from these tests in our cases. It is true that the blood calcium levels were above 12 mg. per 100 cc. of blood in five instances. The serum albumin levels were all normal, the serum globulin levels all slightly to moderately increased."

5 references, 2 tables. (Author's Summary)

Cantor, H. (Petersburg, Va.)

CANCER FOLLOWING BURN SCARS. Virginia M. Monthly 75: 197-199 (Apr.) 1948.

"Carcinoma developing in burn scars are two types: the acute wound cancer occurring in quite superficial burns in older people with atrophic, keratotic skin and chronic scar cancer. Basal cell cancers developing in burn scars usually occur when the burn is superficial, sparing hair follicles and sweat glands. The potentiality of a scar to undergo malignant degeneration and the histological variety of epithelioma resulting are due to the extent of the surface area involved and to the depth of the burn.

The age of the scar is more important than the age of the individual. Cancer of burn scars may occur in younger persons if the cicatrix exists from infancy or childhood."
7 references. (Author's Summary)

Fairburn, E. A.; Burgen, A. S. V. (Middlesex Hosp.)
THE SKIN LESIONS OF MONOCYTIC LEUKAEMIA. Brit. J. Cancer 1:352-362 (Dec.) 1947.

"A case of monocytic leukaemia is described in which other leukaemic symptoms were preceded for several months by a recurrent furunculosis. The patient eventually developed ulceronodular lesions on one arm, which were proved by biopsy to be due to infiltration with monocytic leukaemia.

The clinical and pathological features of the various rashes encountered in monocytic leukaemia are discussed and their evolution postulated." (Author's Summary.)
5 tables, 7 plates, 35 references.

Blue Shield Enrollment

According to the official Enrollment Report issued by the Blue Shield Commission, Arizona Blue Shield had the second largest growth in the United States during the first nine months of 1948. As of the end of 1948, over 33,000 subscribers were enrolled in Arizona Blue Shield.

Experiment in Medical Service

A large Arizona employer, who is paying the entire cost of Blue Shield and Blue Cross for his employees and families, (approximately 400 subscribers) has volunteered to act as an experimental group for the provision of medical service. It is planned to provide medical coverage for "in-hospital" cases on the following indemnity basis: $5.00 for first visit, $3.00 for succeeding visits; limit of one visit per day; for 21 days first year, 25 days second year, 30 days third year and thereafter, plus 90 days at 50% for each admission for a separate illness. There will be some limitations as to what medical illnesses are covered, these to be determined from experience of other Plans. For the present time, it is planned to have the subscriber notify the Blue Shield Office and have the "Statement of Service" go out directly to the Participating Physician. After return of Statement, payments will be made directly to the Participating Physician. Please note that this is an experimental group and the employer is assuming any financial responsibility which may be involved. Incidentally, this experiment was undertaken with the approval of the Professional and Executive Committees.

The Subscriber Insists!!!

If in your opinion, a patient of yours is not eligible for Blue Shield coverage, *but insists* on having you send in a Statement of Service—send it in. *However, please attach a note* giving your opinion in regard to ineligibility, as this will enable the Medical Director to take immediate action. We feel this procedure will tend to eliminate friction between Doctor and subscriber and place the responsibility for rejection of coverage on Blue Shield. The *success* of this procedure will depend entirely on your attaching the note of explanation to the Statement of Service.

The Professional Board of the Arizona Medical Association, as a part of its program for postgraduate medical training, has asked the hospitals of Phoenix and Tucson to send announcements of their scientific staff meetings to the doctors in the surrounding towns and counties. All doctors who receive notices will be welcome to the meeting as the hospitals frequently have excellent scientific programs, including out-of-town speakers.

The Arizona Pediatric Society will be addressel by Dr. Waldo Nelson, Professor of Pediatrics, Temple University, Philadelphia, late in February. The meeting is planned for Phoenix. Any doctors interested in pediatrics and wishing to attend or to become a member of the Arizona Pediatrics Society, will please write Dr. Hugh C. Thompson, 110 South Scott Street, Tucson, Arizona. The Society will also have a clinical day immediately preceding the meeting of the Arizona Medical Association in May. It is planned to have this clinical day conducted by Dr. Harry Ebbs, Associate Professor of Pediatrics, Toronto University. The meeting will be open to any doctor interested in pediatrics. Those desiring to attend should notify Dr. Thompson.

Selected Abstracts

ANTICOAGULANTS

Report of the Committee for the Evaluation of Anticoagulants in the Treatment of Coronary Thrombosis with Myocardial infarctions. Irving S. Wright, M. D.; Charles D. Marple, M. D.; and Dorothy Fahs Beck, Ph. D., New York, New York. Amer. Heart Jr., 36: 801-816, Dec. 1948.

In the spring of 1946, the Board of Directors of the American Heart Association authorized the formation of a Committee for the evaluation of Anticoagulants in the Treatment of Coronary Thrombosis with Myocardial Infarction. This study is being conducted by a group of internists in sixteen hospitals. This report is based on the data obtained from the study of the first 800 cases reported to the Central Laboratory. Three hundred sixty-eight patients admitted on "even" days received conventional therapy and constitute "the control group." Four hundred thirty-two patients admitted on "odd" days received anticoagulants in addition to the conventional treatment and are the "treated group."

The principles used as guides were as follows:

a. Heparin may be given for the first 48 hours or more, if desired.

b. Prothrombin determinations are to be done each day and no Dicumarol should ever be ordered unless the morning prothrombin report is available.

c. Dicumarol, 200 to 300 mg. daily, should be given until the prothrombin time is 30 seconds.

d. Dicumarol, 50 to 100 mg. daily, should be given if the prothrombin time is between 30 and 35 seconds.

e. Dicumarol is withheld if the prothrombin time is 35 seconds or more. Then, no drug is given until the prothrombin time is again down to 30 seconds or less, after which the drug is again given cautiously in 100 mg. doses.

f. The Link-Shapiro technique, using undiluted whole plasma, or the Quick method is to be used for determining the prothrombin time and it is suggested that the Link-Shapiro, using 12.5 per cent diluted plasma, be employed as an additional check or safeguard.

g. Unless contraindications arise, the Dicumarol therapy is to be continued over a minimum period of 30 days; preferably thirty days after the last thromboembolic episode.

h. In instances of hemorrhagic manifestations, the use of synthetic vitamin K preparations in doses of 60 to 75 mg. and transfusion of fresh whole blood (may be citrated) are recommended.

The death rates and the incidence of thromboembolic complications are reported for the control and treated groups as a whole, by week of illness, by age of the patient, and by type and location of the specific thromboembolic complication. Twenty-four per cent of the control patients died, whereas only 15 per cent of the treated group died. As anticipated, anticoagulants reduced the death rate largely by reducing the incidence of the thromboembolic

IMPORTANT NOTICE

The Staff of the Lois Grunow Memorial Clinic is presenting its Third Annual Lecture Series in Medical Sciences at the Lois Grunow Memorial Clinic, 926 E. McDowell Road, Phoenix, Arizona on February 24, 25 and 26, 1949.

The guest speakers will include:

Dr. John C. Jones, Assistant Professor of Surgery (Thoracic), University of Southern California, Los Angeles, California

Dr. Kinsey Macleod Simonton, Assistant Professor of Otolaryngology, Mayo Clinic, Rochester, Minnesota

Dr. A. G. Bower, Clinical Professor of Medicine, Communicable Diseases, University of Southern California, Los Angeles, California

Dr. Joseph Boyes, Plastic Surgery of the Hand, Los Angeles, California

complications. The data indicate that anticoagulant therapy, if not used before, should be begun as late as the second or third week after a myocardial infarction, or later if complications have developed, and that the therapy should be continued for at least four weeks after the last thromboembolic episode.

Thromboembolic complications were recognized, clinically, in 36 of each 100 "control" cases, and in 14 of each 100 treated cases. This protection from these complications is further emphasized by the report that there were only 6.5 per 100 such complications in cases which were under full therapeutic effect of the drug, five of which occurred while the patients were not receiving the drug. There was evidence of extension of the original thrombosis in nine cases per 100 in the control series as against two cases per 100 in the treated group. There was infarction of a new area in the myocardium in 6.5 cases per 100 in the control patients as against 2.5 among the treated patients.

Hemorrhagic manifestations occurred in 6 per cent of the control group and in 12 per cent of those treated with anticoagulant therapy. However, five of the latter were not due to or aggravated by the anticoagulants. Seven per cent of the treated group had hemorrhages which were due to or aggravated by the anticoagulant drugs, and only three per cent of these hemorrhages were classed as severe. In the absence of other hemorrhagic states, the hazards from hemorrhage due to anticoagulants are not sufficient to contraindicate their use, provided there are facilities for adequate laboratory and clinical control.

It is concluded from their study that anticoagulant therapy should be used in all cases of coronary thrombosis with myocardial infarction unless a definite contraindication exists.

Leslie B. Smith, M. D.

HEMOPTYSIS

Diseases of the Chest. El Paso, Texas. 15: 824-842 (Nov. - Dec.), 1948.

The Clinical Significance of Pulmonary Hemorrhage: A Study of 1316 Patients with Chest Diseases. Osser A. Abbott, M. D.

Dr. Abbott has analyzed and determined the cause of hemoptysis in 497 cases which were seen by him. Frank hemoptysis was present in 318 patients, scant or streaking of blood was present in 174 cases, and the degree was unknown in five patients. The incidence of bleeding in each of the 50 diseases is given, and for the 12 most frequent diseases was as follows:

Bronchogenic Carcinoma	53.6 per cent
Lung abscess	49.2 " "
Pulmonary infarct	44.0 " "
Bronchiectasis	43.5 " "
Tuberculosis	36.5 " "
Congenital Cyst	25.8 " "
Empyema	24.5 " "
Metastatic carcinoma	24.0 " "
Mediastinal tumor	20.0 " "
Cardiac disease	17.5 " "
Esophageal obstruction	9.0 " "

The author found that there is a distinct tendency for the symptom of pulmonary bleeding to occur relatively late in the sequence of the symptom in patients with bronchogenic carcinoma.

The symptom of bleeding was associated with serious disease in 90 per cent of the cases. He stresses the relatively higher incidence of pulmonary bleeding in cases of bronchogenic carcinoma, bronchiectasis and lung abscess than in pulmonary tuberculosis. There is an absence of correlation between the type of bleeding and the underlying disease. He emphasizes the need for a complete study of all patients who present the symptom of bleeding before making a diagnosis or deciding that the cause is undetermined.

Leslie B. Smith, M. D.

SUPERIOR WEIGHT GAINS IN PREMATURES

When 100 premature infants were given an enzymic casein hydrolysate and dextrose mixed with breast milk, the infants tolerated the mixture well. There were no signs of digestive disturbance. In every instance, infants receiving the supplement showed a greater gain in weight than did infants receiving only breast milk.* Protolysate, Mead Johnson & Company's enzymic digest of casein, is effective when used as a supplement to breast milk for premature infants.

For literature and professional samples of protolysate, write Mead Johnson & Company, Evansville 21, Indiana.

*Jorpes, J. E.: Magnusson, J. H., and Wretlind, A.: Casein Hydrolysate: A Supplementary Food for Premature Infants: Lancet 251:228-232 (Aug. 17) 1946.

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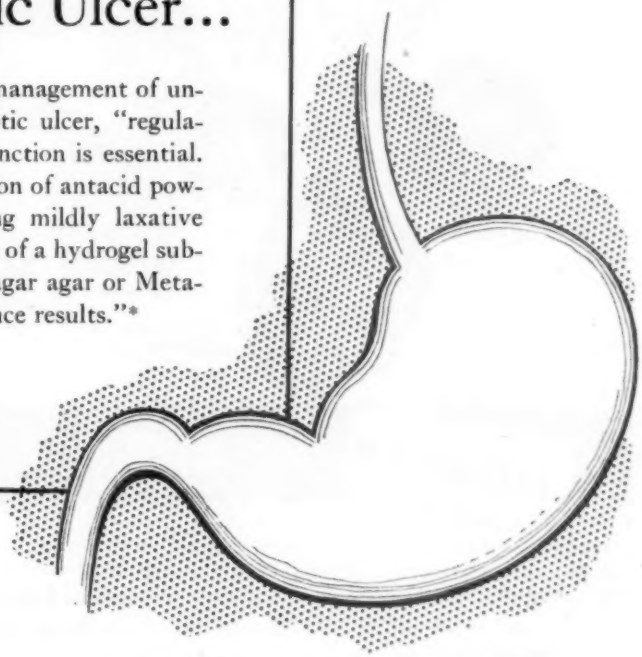

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*Gerendasy, J.: Modern Treatment of Peptic Ulcer, J. M. Soc. New Jersey *43*:84 (March) 1946.

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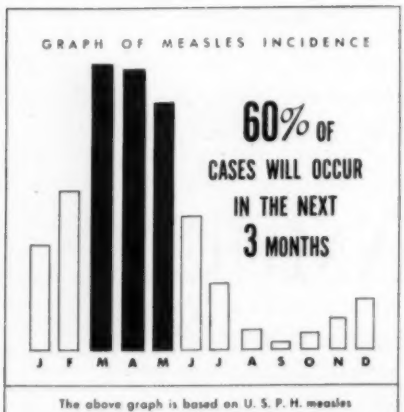

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Journal of

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Vol. 6 February, 1949 No. 2

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Editorials

The A.M.A. Special Assessment

Federal agencies in Washington continue to grind out propaganda advocating compulsory health insurance. Whenever Federal Security Administrator Oscar Ewing, Representative Dingell or Senator Murray speak they are given big headlines. On the other hand the program authorized by the House of Delegates of the A.M.A. at the St. Louis session is rapidly taknig form. The $25 special assessment levied by the Board of Trustees on all members of the Association has been widely approved.

It is estimated to raise $3,500,000. When Mr. Ewing was first informed of this action by the medical profession he called it a "three and a half million dollar lobby fund." Mr. Dingell stated that half the doctors would refuse to pay it. If the doctors of this country have any pride, Mr. Dingell's remarks should be worth at least a million dollars.

It must be pointed out that the American Medical Association can not violate the provisions of its charter by setting up a Washington lobby to oppose socialized medicine in Congress. The objects of this special assessment are: to enlarge the Washington office and place it directly under the Board of Trustees; to secure suitable public relations counsel; to acquaint the people with the superior advantages of the American practice of medicine over the government dominated systems of other countries; and to point out the dangers of a politically controlled compulsory health insurance plan. It will also advertise the voluntary prepaid insurance plans.

The first step in the implementation of this program was made by the Board of Trustees in setting up a Planning Committee. This will consist of ten members; four from the Board of Trustees and officers and three from the House of Delegates. The other three will be the President, the Chairman of the Board, and the Secretary of the Association. As a result the Planning Commission will consist of:

Drs. Edwin S. Hamilton, Illinois; Gunnar Gundersen, Wisconsin; Walter B. Martin, Virginia, and Louis H. Bauer of New York, all members of the Board of Trustees; Drs. William Bates, Pennsylvania; John W. Cline, California, and R. B. Robins, Democratic National Committeeman of Arkansas, all members of the House of Delegates; President R. L. Sensenich, Indiana; Chairman Elmer L. Henderson, Kentucky, and Geo. Lull, Secretary of the A. M. A.

A great deal of organizational work has already been done by this group. At a meeting in A. M. A. headquarters in Chicago on December 8, the executive committee of the Planning Board decided to employ Clem Whitaker and Leone Baxter of the public relations firm of Whitaker & Baxter of San Francisco as public relations counsel to direct the entire campaign. The firm's address is DeYoung Building, San Francisco (4) Calif.

The campaign will be directed from a Chicago and Washington office, working closely with A. M. A. headquarters, with A. M. A.'s public relations department.

The appointment of Clem Whitaker of San Francisco as public relations counsel has the ear marks of a master stroke. In his meteoric rise since becoming Governor of California, Mr. Earl Warren has suffered two defeats. The other one was by the California Medical Association when they defeated the Governor's Bill in the California legislature for compulsory health insurance, not only once but twice. California physicians may point with pride to this accomplishment. And Clem Whitaker was the guiding hand during both battles.

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from the vast number of the medical profession that the decision by the A.M.A. to carry on its own fight against socialized medicine in a real militant campaign is an action long over due, occasional rumblings are heard and we have received one letter, which has the evidence of considerable circulation, that the doctors should find out how the money is going to be spent before it is contributed. Such a letter must be a great consolation to Mr. Ewing and Mr. Murray. It reminds one of the great late Will Rogers when he commented on certain individualists who refused to play ball with the government in the big depression, unless they could see the next card on the deck before it was played.

The decision by the A. M. A. House of Delegates in St. Louis was not an "on the spur of the moment" action. It represented the climax of a debate which has been going on for at least 15 years. And when the House finally decided to take action it was by unanimous acclaim. It has been ascertained by the Office of the A. M. A. in Chicago that to date about 1500 members of the Association have given their time and financial support in the battle of medical economics. Now with every member of the organization being given an opportunity to contribute directly, it is much more likely that he will take an interest in how the campaign will be conducted and will carry on his own part. It must not be forgotten that the contribution of $25 will be the only participation necessary. It will take much more money, at both the National and State levels, and even at the county levels to defeat the totalitarian forces who are trying to destroy free enterprise in this nation by first socializing the private practice of medicine. The program so far outlined by the A. M. A. will lay the ground work only by educating the great masses of the population. The final battle will be won by each physician in the nation who believes in this program, by persuading his patients, and friends, and all the public to write to or contact their Representative in the Congress in Washington to oppose all measures pertaining to compulsory health insurance.

Topical Use of Sulfonamides and Penicillin Decried

Attention is directed to the opinions of Sulzberger and Baer[1] concerning the wisdom of the topical application of sulfonamides and anti-

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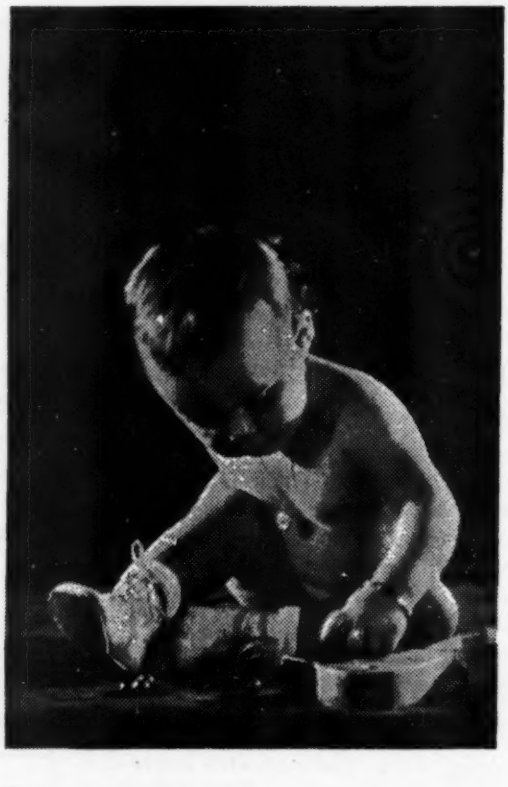

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biotics. The criteria of a suitable topical agent, they state, are:

1. It should have greater therapeutic effectiveness than other medicaments which might be used in the particular case at hand.

2. It should have a lower sensitizing potential, lower irritancy, and fewer systemic side-effects than other medicaments which might be used for the same purposes.

3. It should be an agent which is not administered systemically and would not therefore present the possibility that topical use in the prevention or treatment of a skin disease might sensitize the individual and interfere with subsequent administration of the medicament when it may be urgently required for prevention or treatment of general or visceral disease.

With these criteria in mind Sulzberger and Baer decry the topical use of sulfonamides because:

1. Except in rare instances topical application of sulfonamides is not significantly more effective than that of most older and certain newer antibacterial agents.

2. The sulfonamides have a sensitizing potential equal to or greater than that of most older and some newer antibacterial agents which can be used topically. Furthermore they have greater systemic by-effects than many other available products.

3. The sulfonamides still have an important place in the treatment of certain general infections and serious diseases. Obviously, therefore, prior sensitization by topical application of sulfonamides may seriously interfere with later administration of these drugs when they are urgently needed.

It is concluded, therefore, that sulfonamides are not suitable drugs for general topical use in diseases of the skin.

The authors, using exactly the same line of reasoning with regard to penicillin, arrive at the same conclusion, namely that penicillin is not a suitable agent to be employed topically for general use against bacterial infections of the skin.

Tyrothricin, on the other hand, being highly effective, having a low sensitizing potential, and having no systemic uses, is a suitable and very useful antibacterial agent for topical use in

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SPECIAL NOTICE

Speakers for the third ANNUAL LECTURES IN MEDICAL SCIENCES to be presented by the Staff of The Lois Grunow Memorial Clinic on February 24, 25, and 26, 1949:

Dr. John C. Jones	1. Surgical Aspect of Congenital Heart Lesions
	2. Thoracic Problem of the Infant and Child
	3. Treatment of Acute and Chronic Pulmonary Suppuration
Dr. Kinsey Macleod Simonton	1. Symptoms of Dizziness
	2. Treatment of Paranasal Sinusitis
	3. Treatment of Nasal Polyps
Dr. A. G. Bower	1. Brucellosis
	2. Q Fever
	3. Infectious Mononucleosis
Dr. Joseph Boyes	1. Immediate Care of Injury of the Hand with Special Reference to Factors Determining the Time of Repair of the Flexor Tendon
	2. Tendon Grafts and Tendon Transplants in the Hand.
	3. Sound movie: Early Care of Wounds of the Hand.
Dr. W. Warner Watkins	1. Differential Diagnosis of Multiple Nodular Shadows in Lungs
Dr. Paul S. Causey	1. Saddle Block Anesthesia and Obstetrics
Dr. L. L. Stolfa	1. Renal Insufficiencies
Dr. Stanford F. Hartman	1. Modern Concepts of Cerebral Palsy

skin infections. For similar reasons bacitracin probably will likewise be found to be useful.[2]

1. Sulzberger, M. B., and Baer, R. L.: The 1946 Year Book of Dermatology and Syphilology. Chicago, Year Book Publishers, Inc., 1947. Pp. 8, 11, 14.

2. The statement concerning bacitracin is not the expressed opinion of Sulzberger and Baer, but rather that of the present editorial writer.

Personal Notes

DR. HOLLIS H. BRAINARD, who has been associated with the Thomas-Davis Clinic in Tucson as a gynecologist and obstetrician, has resigned to open an office at 2440 East Sixth Street.

DR. HILDA H. KROEGER has been appointed administrative assistant to the director of the Grace-New Haven Community Hospital in Connecticut. Dr. Kroeger has just completed the course in hospital administration at Yale. She was educated at the Universities of Arizona and Illinois, obtained her M. D. degree at Rush, was a member of the Tucson City-County health department, and was director of maternal and child health for the State of Arizona.

The Apache-Navajo Medical Society elected **DR. ARNOLD H. DYSTERHEFT** of McNary its president; **DR. FRANK A. COCUZZI,** Showlow, vice-president; **DR. ELLIS V. BROWNING,** Springerville, Secretary and **DR. GEORGE HANDALL,** St. Johns, Treasurer. The society was organized to aid the public in an educational program to increase health standards and child welfare.

DR. OSCAR W. FRISKE, formerly resident in Surgery at the Good Samaritan Hospital, Phoenix, then in practice in Safford, Arizona, has established an office in Douglas, Arizona.

The Pima County Council of Social Agencies is planning a survey of Tucson health conditions. Among the members of the health section are **DR. R. W. RUDOLPH,** Chairman, and **DR. L. H. HOWARD.**

DR. GEORGE m. de YOUNG, of 2613 East Helen Street, Tucson, was badly injured in December, when his car left the road on "A" mountain and rolled down the hillside.

The report by **DR. W. H. OATWAY** on the use of routine chest x-raying in general hospitals of the United States, published in the January issue of ARIZONA MEDICINE, has been selected as subject for a monthly abstract of the Na-

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tional Tuberculosis Association. About 60,000 abstracts are sent out to physicians, and several thousand reprints of the article are used in case-finding exhibits. DR. OATWAY, on leave from his practice in Tucson, is reported improving at Barlow Sanatorium in Los Angeles.

The new Superintendent of the Pima County General Hospital, and also county physician, is **DR. FRANCIS J. BEAN,** who formally took the positions on November 15th. He replaces **DR. JAMES F. TERRELL,** who resigned in May, 1948 to return to active duty with the U. S. Navy, and who is presently at San Diego.

DR. PAUL B. JARRETT, Phoenix, has moved his office from 801 North First Avenue to 543 East McDowell Road.

The Tucson Medical Center has announced the membership of its various governing groups for the coming year; the **REV. GEORGE FERGUSON** was re-elected president of the Board of Directors. A citizens' advisory board of 100 persons has recently been appointed. The medical staff officers now include **DR. ROY HEWITT,** president; **DR. J. B. LITTLEFIELD,** vice-president; **DR. JACKMAN PYRE,** secretary; and **DRS. CLARENCE ROBBINS, CHARLES NEWCOMB, O. J. FARNESS** and **WILLIAM MANNING,** advisory board members. **MR. CLYDE FOX** is the hospital administrator.

DR. GEORGE DIXON, orthopedic surgeon of Tucson, has arranged for the construction of a building on Country Club Road which will house his offices and facilities for a physiotherapist.

DR. HENRY J. STEFFENS, Phoenix, suffered minor injuries in a car accident recently.

DR. HOWARD D. COGSWELL of Tucson presented a paper entitled "Hidden Carcinoma of the Breast" at a meeting of the Western Surgical Association in St. Louis in December, 1948.

The staff of the Arizona State Department of Health is now composed of **DR. JAMES P. WARD,** who replaced **DR. GEORGE MANNING** as Director; and **DR. ELINORE P. KALBAUGH,** who has recently replaced **DR. GERALD R. CLARK** as Director of The Tuberculosis Control Division, and as acting-director of the Venereal Disease Control Divisions. **MR. FRANK WILLIAMS** is Director of the Health Education Division.

The State Board of Health has adopted a reorganization plan for the department. It includes the establishment of several bureaus, for which new personnel are to be obtained by funds from a budget which has just been submitted to the legislature. The bureaus will be concerned with administration, preventive medical services, local health services and sanitation.

Comstock Children's Hospital of Tucson, operated by Community Service, Inc., has installed a new Director, MRS. AILEEN M. TAYLOR. The Tuberculosis Section, which cares for fifteen children, is in charge of **DR. E. UOQODA,** with **DR. COCHRANE** as consulting pediatrician. The Crippled Children Section, which also contains

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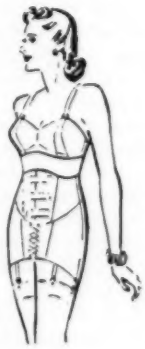

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fifteen children, is cared for by a current staff of **DRS. JAMES SICKLER, JOHN SCHWARTZMANN, GEORGE DIXON** and **R. E. HASTINGS.**

The January meeting of the Medical Staff of the Tucson Medical Center was presented with a "Symposium on Acute Infectious Hepatitis" by **DRS. M. CARREROS, HARRIET BARITALL,** and **MALCOLM DENLEY. DR. DENLEY** is the current Medical Resident at the hospital.

DR. NORMAN HALL, Phoenix, has been appointed Maricopa County physician, following the resignation of **DR. FRANK EDEL.**

DR. DERMONT MELICK, Phoenix, has organized a journal club for surgery, to meet the 2nd and 4th Thursdays of the month at St. Joseph's Hospital, Phoenix. **DR. HAYES CALDWELL,** Phoenix, has organized a similar journal club for medicine to meet on the 1st and 3rd Thursdays.

DR. JOHN SCARFF, Clinical Professor of Neurological Surgery at Columbia University, addressed the Pima County Medical Society in January on "Unilateral Pre-Frontal Lobotomy for the Relief of Intractable Pain and the Termination of Drug Addiction."

DR. E. W. PHILLIPS, retired Phoenix physician, spoke at the January Maricopa County Medical Society Meeting on "Allergy to Pituitary Gonadotropins."

DR. KENNETH C. BAKER announces the opening of his new offices on January 3, 1949 at 729 North Fourth Avenue, Tucson, Arizona.

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Book Reviews

PREOPERATIVE AND POSTOPERATIVE CARE OF SURGICAL PATIENTS. By Hugh C. Ilgenfritz. Cloth. Price $10.00. Pp. 858, with 110 illustrations. The C. V. Mosby Company. 3207 Washington Blvd., St. Louis 3, Missouri. 1948.

This volume is an excellent and thorough presentation of the factors upon which success in surgical treatment depends. Roughly, the first half of the book deals with the general features in the preparation and follow-up care of patients. In this portion the essential relation of physiology, chemistry and pathology to the treatment is explained, and such general topics as sedative medication, shock, chemotherapy, transfusion, and the association of organic disease and systemic factors are covered.

After a discussion of minor and major postoperative complications, the second half of the book dwells on specific anatomical areas and surgical conditions. While there is some repetition of the material in the first half of the book, it is considered justified, and this arrangement makes the book usable as a reference for a rapid

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Woman's Auxiliary

MARICOPA COUNTY NEWS

In December the Auxiliary to the Maricopa County Medical Society held its annual Christmas, formal Dinner Dance at the Westward Ho Hotel in the Fiesta Room. This party took the place of the regular December meeting of the Auxiliary. The affair was handled by Mrs. Charles VanEpps, who deserves a great deal of credit for its success.

The January meeting was at the home of Mrs. Charles Ploussard. Mr. J. Thomas McIntire, Superintendent of the Arizona Children's Colony, spoke on the survey which has been done among the mentally deficient children and of the proposals which are to be made to the 19th Legislature for the establishment of the colony and the appropriation of the necessary funds. It was voted that the Auxiliary should support the bill concerning the Children's Colony and urge its passage. Mrs. Kober, Legislative Chairman, is contacting the membership, asking for their personal support and their appeal to their legislators.

Isabella Williamson,
Maricopa Publicity Chairman.

PIMA COUNTY

Members of Pima County Woman's Auxiliary met in regular session Tuesday night, January 11, 1949 at the home of Dr. and Mrs. Storts.

Speaker was Julia Fuller. She talked on Child Colony and suggested Pima County Auxiliary members pass a resolution favoring Arizona Children's Colony Bill. This resolution was passed.

The State President, Mrs. Thomas Bate and the State Treasurer, Mrs. Karl Harris attended this meeting as representatives of the Arizona State Medical Auxiliary.

Respectfully submitted,
Mrs. H. Kohl, Tucson.

BIOGRAPHICAL SKETCH OF PUBLIC HEALTH CHAIRMAN, MRS. O. W. THOENY

Dorothy S. Thoeny (Mrs. Oscar W. Thoeny), chairman of Public Health committee of the Woman's Auxiliary to the Arizona Medical Association, was born in Nebraska and graduated from the University of Wisconsin with an A. B. (course in Journalism) degree. A charter mem-

ber of the Woman's Auxiliary to the Arizona Medical Association, she has also served it as Corresponding Secretary in the Presidency of Mrs. C. R. Swackhamer, Superior, Arizona. She was Recording Secretary of the State Auxiliary in 1936 and was also president of the Woman's Auxiliary to Maricopa County Medical Association in 1938-39.

She was married to Dr. Oscar W. Thoeny in Nebraska on June 27, 1927, and they lived in San Diego during the time of his internship at the U. S. Naval Hospital there. In 1929, following his naval service, they came to Phoenix, where they have resided except for a four-year interim in El Paso, Texas in the Army Medical Corps during World War II.

Respectfully submitted,

Mona Cohen,

State Publicity Chairman.

Mrs. Oscar W. Thoeny

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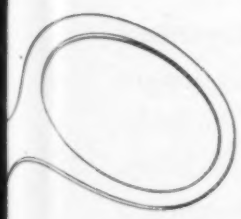

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[1] McLester, J. S.: Nutrition and Diet in Health and Disease, ed. 4, Philadelphia and London, W. B. Saunders Company, 1943.

[2] Kunde, M. M.: The Role of Hormones in the Treatment of Obesity, Ann. Int. Med. *28*:971 (May) 1948.

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